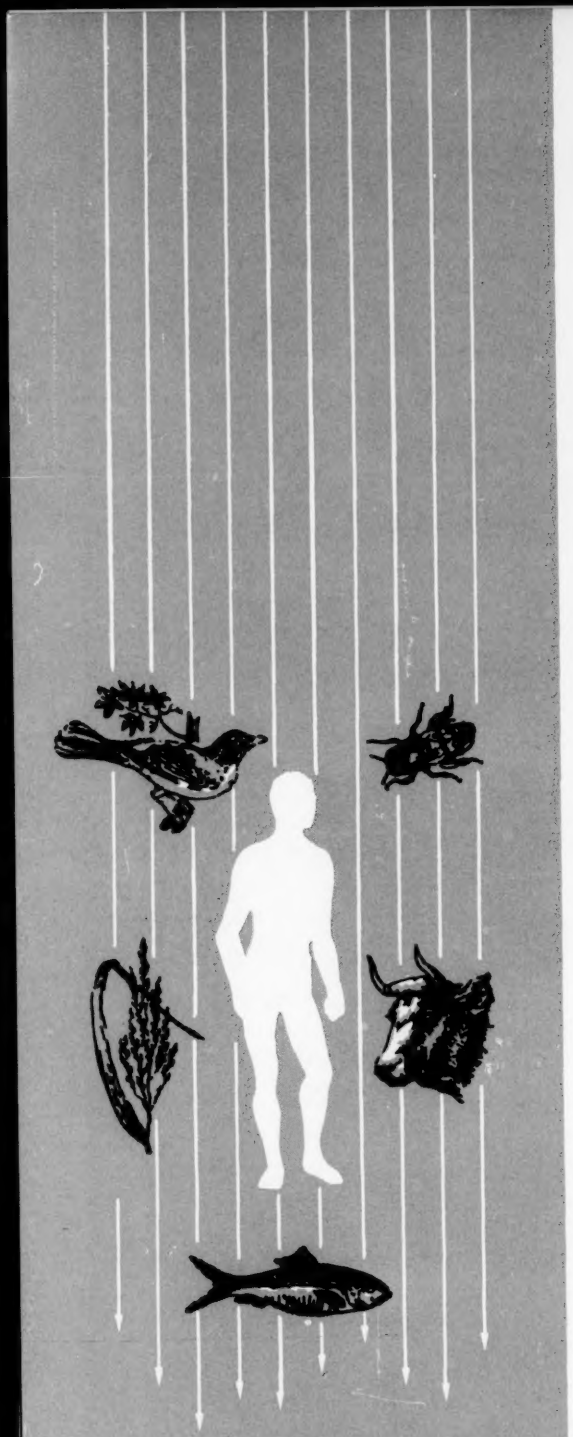

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The Monitoring Panel consists of representatives of the Agricultural Research Service, Animal and Plant Health Inspection Service, Extension Service, Forest Service, Department of Defense, Fish and Wildlife Service, Geological Survey, Food and Drug Administration, Environmental Protection Agency, National Marine Fisheries Service, National Science Foundation, and Tennessee Valley Authority.

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FISH, WILDLIFE, AND ESTUARIES

Organochlorine Pollutants in Small Cetaceans from the Pacific and South Atlantic Oceans, November 1968–June 1976[1]

Thomas J. O'Shea,[2] Robert L. Brownell, Jr.,[3] Donald R. Clark, Jr.,[3] William A. Walker,[4] Martha L. Gay,[2] and Thair G. Lamont[3]

ABSTRACT

Organochlorine residues were analyzed in blubber, brain, or muscle tissues of 69 individuals representing 10 species of small cetaceans. Collections were made from November 1968 through June 1976 at localities in the Eastern Tropical Pacific and along the coasts of California, Hawaii, Japan, and Uruguay. Relations of residue concentrations between tissues are described for DDE and PCBs in two dolphin species. ΣDDT and PCB residues in blubber of most of the 19 individuals of the five southern California species sampled exceed concentrations that are associated with reproductive impairment in pinnipeds, although the nature of such associations is not well defined. The ΣDDT residue of 2,695 ppm in blubber of one California coastal Tursiops truncatus *is one of the highest concentrations reported in tissues of members of any population of wild mammals. Except for one rough-toothed dolphin* (Steno bredanensis) *from Maui, Hawaii, all individuals from all localities surveyed were contaminated with organochlorine compounds. Seventeen different organochlorines were detected; greatest diversity occurred near Japan and California. This is the first report of several of these compounds in tissues of any species of marine mammals. The o,p'-isomers and metabolites of DDT were detected unusually frequently. Ratios of p,p'-DDT to p,p'-DDE in blubber of cetaceans from waters off countries where use of this pesticide has been relatively recent and ongoing were at least an order of magnitude higher than in cetaceans from United States waters.*

Introduction

The status of marine mammal populations is a subject of much recent interest and concern. Legislation, particularly the U.S. Marine Mammal Protection Act of 1972, and international agreements have been formulated with the intention of safeguarding these populations. It is of continuing importance, therefore, to monitor organochlorine pollutants in tissues of these species, many of which occupy high trophic levels in marine food webs.

Elevated concentrations of pesticides and polychlorinated biphenyls (PCBs) are known to occur in blubber of cetaceans (*1, 15, 23*) and pinnipeds (*14, 18*) in geographical areas subject to contamination with these compounds. The present study represents a survey of organochlorine residues in tissues of 69 individuals of 10 species of small cetaceans collected at one South Atlantic and several Pacific localities from November 1968 through June 1976. These localities have had contrasting histories of exposure to organochlorines, and with the exception of two individual specimens from California (*23*), small cetaceans from these areas have not been previously surveyed for residue concentrations. Lack of such information is due partly to the scarcity and difficulty in obtaining adequate specimens suitable for analysis.

Sampling and Analyses

Tissues were obtained from beach-stranded animals or from free-ranging individuals captured by fishermen. All samples from Hawaii, southern California, and Tokyo Bay were taken from beach-stranded animals, except for two pilot whales (*Globicephala macrorhynchus*) which were caught at sea off Los Angeles County, California. Specimens from the remaining localities were taken at sea. Tissues sampled were blubber, brain, and muscle (*longissimus dorsi*). All dissections were performed by either of the same two investigators, and tissue samples were always taken from the same topo-

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graphic locations on each specimen. All specimens were preserved in 10 percent formalin. However, due to logistical problems inherent in obtaining tissues from marine mammals, the initial methods of preservation varied with the field situation. Specimens from Uruguay and Japan, except finless porpoises (*Neophocaena phocaenoides*), were immediately preserved in formalin. All California specimens, except two coastal bottlenose dolphins (*Tursiops truncatus*) held frozen for 12 and 18 days and five common dolphins (*Delphinus delphis*) frozen for 1–8 months, were also preserved in formalin on dissection. Other specimens were frozen for various lengths of time before transfer to formalin. Hawaiian dolphins were frozen for 1 month, specimens from the Eastern Tropical Pacific for 2–36 months, and Japanese finless porpoises for 4–57 months. All tissues preserved in formalin were stored in glass jars rinsed with acetone and hexane. Lids were lined with Teflon or aluminum foil. Twenty-five samples of the original preservative stored in similar jars were extracted with hexane and analyzed for organochlorine compounds by gas-liquid chromatography; none was detected.

Collection took place in the western South Atlantic off Uruguay during November–December 1974 and in the Eastern Tropical Pacific between April 1973 and February 1976; all California animals were sampled between May 1974 and June 1976. The Hawaiian strandings occurred in June 1976. Samples from Japan were taken between November 1968 and February 1975. External measurements and notes on stomach contents, parasites, relative age, and reproductive condition were obtained for some of the specimens but are not reported here. These data, as well as precise locations of California stranding sites and specific dates, are available from the authors.

Tissues were removed from formalin and patted dry before being weighed. Residues are expressed as wet weight of preserved tissue. A 10-g portion of homogenized tissue was mixed with anhydrous sodium sulfate and extracted for 7 hours with hexane in a Soxhlet apparatus. An aliquot of the hexane extract, equivalent to a 5-g subsample, 2-g equivalent subsample for blubber, was cleaned on a Florisil column. A portion of the eluate was separated into three fractions on a SilicAR column (*5*). The pesticides in each fraction were identified and quantitated with a gas-liquid chromatograph equipped with an electron-capture detector and a column packed with a mixture of 1.5 percent OV–17 and 1.95 percent QF–1. An OV–275 column was used to confirm and/or quantitate *o,p'*-TDE, *o,p'*-DDT, and *o,p'*-DDE. In some samples where both *o,p'*-DDE and *p,p'*DDE overlapped into fraction III, *cis*-chlordane could not be quantitated. PCBs were estimated by comparing total area of PCB peaks with that of Aroclor 1254 or 1260, depending on which profile the sample more closely resembled. The average recoveries from spiked tissues ranged from 80 percent to 104 percent. Residues were not corrected on the basis of these data.

The lower limit of sensitivity was 0.1 ppm for organochlorines and 0.5 ppm for PCBs. Residues in about 10 percent of the samples were confirmed on a gas chromatograph/mass spectrometer. Residue data are reported by locality, species, sex, tissue, and total body length (an indication of relative age) for each animal. In this way the reader may compute statistics as desired from the original data. Geometric means and 95 percent confidence limits are provided for sample sizes of five or greater. Not-detected values were considered zero in computations; geometric means were therefore calculated on data transformed as $\ln(x + 1)$, with 1 subtracted from the retransformed means.

Results

RESIDUES DETECTED

The principal organochlorine residues detected were PCBs and isomers and metabolites of DDT. Residues of *p,p'*-DDT or its metabolites were detected in all but 1 of the 69 individuals sampled. In addition, *o,p'*-isomers of these compounds, seldom reported in tissue samples of warm-blooded animals from high trophic levels, were also found with unusual frequency (Table 1). One or more of the *o,p'*-isomers were detected in representatives of each species from each locality, except one striped dolphin (*Stenella coeruleoalba*) taken at 7° 0'N, 86° 30'W. Residues of *o,p'*-DDE ranged up to 70 ppm in blubber of common dolphins stranded on southern California beaches. PCBs were also detected in each species at every locality, except for the striped dolphin noted above.

Residues of other organochlorines were present at varying frequencies and concentrations (Table 2). Dieldrin occurred in the blubber of all species except the Fraser's dolphin (*Lagenodelphis hosei*), but not in each species at every locality. Maximum concentrations (up to 38 ppm) were reached in the blubber of finless porpoises from coastal Japan. Hexachlorobenzene (HCB), heptachlor epoxide, and components and metabolites of chlordane also occurred in many samples, but usually at levels below 1.0 ppm (Table 2). Toxaphene was detected in blubber of four species of cetaceans from waters off Japan and in striped dolphins collected at 9° 41'N, 98° 7'W. Endrin, mirex, and *cis*-nonachlor residues (not tabulated) were detected infrequently. Endrin occurred at 0.22 ppm in blubber of a male striped dolphin taken at sea at 9° 41'N, 98° 7'W and at 0.24 ppm in blubber of a male common dolphin stranded in Tokyo Bay. Mirex was found at 0.18 ppm

TABLE 1. *Residues of the principal organochlorine compounds (isomers and metabolites of DDT and PCBs) found in blubber, brain, and muscle tissues of small cetaceans from the Pacific and South Atlantic Oceans, November 1968–June 1976*

Locality and Species	Sex	Tissue (N)	TL	*p,p'*-DDE	*p,p'*-TDE	*p,p'*-DDT	*o,p'*-DDE	*o,p'*-TDE	*o,p'*-DDT	PCBs
URUGUAY										
34°22′S, 53°46′W										
Franciscana dolphin	F	Blubber (3)	116	2.4	2.4	3.0	ND	7.4	0.56	3.6
			133	2.2	1.2	5.6	ND	ND	0.28	4.0
			138	1.9	0.86	4.2	1.7	7.6	7.6	4.2
		Brain (3)	116	ND	ND	ND	ND	ND	ND	0.35
			133	ND	ND	ND	ND	ND	ND	ND
			138	ND	ND	ND	ND	ND	ND	ND
		Muscle (3)	116	ND	ND	ND	ND	ND	ND	0.25
			133	ND	ND	ND	ND	ND	ND	0.13
			138	ND	ND	ND	ND	ND	ND	ND
	M	Blubber (5)	114	5.0	2.8	6.6	0.14	ND	1.4	18
			116	2.6	4.0	10	1.5	11	1.8	3.2
			130	4.4	2.0	7.0	0.10	ND	0.58	5.8
			140	6.4	2.2	7.6	ND	10	1.3	6.8
			145	4.0	1.8	7.2	1.4	4.8	1.1	5.6
			GM	4.3	2.5	7.6	0.50	2.8	1.2	6.8
			CL	2.8–6.4	1.6–3.6	6.2–9.3	ND–1.6	ND–17	0.7–1.8	2.9–14
		Brain (5)	114	ND	ND	ND	ND	ND	ND	ND
			116	ND	ND	ND	ND	ND	ND	ND
			130	ND	ND	ND	ND	ND	ND	0.65
			140	ND	ND	ND	ND	ND	ND	ND
			145	ND	ND	ND	ND	ND	ND	ND
			GM	ND	ND	ND	ND	ND	ND	0.11
			CL							ND–0.46
		Muscle (5)	CL	ND	ND	ND	ND	ND	ND	ND
EASTERN TROPICAL PACIFIC										
3°26′S, 91°41′W										
Fraser's dolphin	M	Blubber (1)	168	7.2	0.72	1.8	ND	1.3	ND	5.2
		Muscle (1)	168	0.17	ND	ND	ND	ND	ND	ND
4°50′S, 88°12′W										
Striped dolphin	F	Blubber (2)	186	1.6	0.28	0.76	ND	0.74	0.18	ND
			187	4.4	0.26	0.88	ND	ND	ND	ND
		Brain (2)	187	ND	ND	ND	ND	ND	ND	ND
		Muscle (2)	187	ND	ND	ND	ND	ND	ND	ND
	M	Blubber (1)	195	2.8	0.40	1.0	ND	1.0	0.44	5.0
		Brain (1)	195	ND	ND	ND	ND	ND	ND	ND
		Muscle (1)	195	ND	ND	ND	ND	ND	ND	ND
7°0′N, 86°30′W										
Striped dolphin	F	Blubber (1)	192	0.60	ND	0.50	ND	ND	ND	ND
		Muscle (1)	192	ND	ND	ND	ND	ND	ND	ND
7°46′N, 84°20′W										
Striped dolphin	F	Blubber (1)	131	76	4.0	34	ND	1.9	3.0	9.8
		Muscle (1)	131	0.55	0.13	ND	ND	ND	ND	ND
9°41′N, 98°7′W										
Striped dolphin	M	Blubber (2)	148	110	6.0	40	1.1	3.0	13	7.6
			160	120	6.4	40	1.1	3.0	12	6.8
		Muscle (2)	148	1.0	0.25	ND	ND	0.12	ND	ND
			160	1.4	0.25	ND	ND	0.13	ND	ND
		Brain (1)	148	1.1	0.16	ND	ND	ND	ND	0.40
17°53′N, 105°6′W										
Striped dolphin	F	Blubber (1)	180	18	2.0	7.4	ND	1.1	1.6	4.4
		Brain (1)	180	0.2	ND	ND	ND	ND	ND	ND
		Muscle (1)	180	0.19	ND	ND	ND	ND	ND	ND
18°25′N, 110°48′W										
Striped dolphin	F	Blubber (1)	194	22	0.92	3.4	ND	0.44	0.58	5.6
		Brain (1)	194	0.22	ND	ND	ND	ND	ND	ND
		Muscle (1)	194	0.12	ND	ND	ND	ND	ND	ND
20°6′N, 110°29′W										
Striped dolphin	F	Blubber (4)	136	6.6	1.1	2.6	ND	0.66	0.46	1.7
			178	19	1.1	3.8	0.10	0.68	1.5	ND
			188	0.46	ND	0.16	ND	ND	ND	ND
			190	18	1.7	4.2	ND	1.1	0.36	12
		Brain (4)	136	0.12	ND	ND	ND	ND	ND	ND
			178	0.20	ND	ND	ND	ND	ND	ND
			188	ND	ND	ND	ND	ND	ND	ND
			190	0.14	ND	ND	ND	ND	ND	ND

TABLE 1. *Continued*

Locality and Species	Sex	Tissue (N)	TL	*p,p'*-DDE	*p,p'*-TDE	*p,p'*-DDT	*o,p'*-DDE	*o,p'*-TDE	*o,p'*-DDT	PCBs
		Muscle (4)	136	0.11	ND	ND	ND	ND	ND	ND
			178	0.25	ND	ND	ND	ND	ND	ND
			188	ND	ND	ND	ND	ND	ND	ND
			190	0.14	ND	ND	ND	ND	ND	ND
	M	Blubber (1)	208	22	0.68	1.6	ND	0.32	0.52	2.6
		Brain (1)	208	0.30	ND	ND	ND	ND	ND	ND
		Muscle (1)	208	0.38	ND	ND	ND	ND	ND	ND
					CALIFORNIA					
32°33′N–35°12′N										
Common dolphin	F	Blubber (3)	182	730	22	21	28	6.8	15	80
			192	360	26	19	26	7.4	12	100
			210	950	51	16	43	8.0	10	160
		Brain (2)	179	20	0.40	0.21	0.39	ND	ND	0.65
			192	14	0.65	ND	0.70	ND	ND	2.2
		Muscle (3)	182	5.5	0.17	ND	0.24	0.12	ND	0.60
			192	4.3	0.28	ND	0.20	ND	ND	0.70
			210	13	0.78	ND	6.8	0.20	ND	3.3
	M	Blubber (10)	184	980	32	26	36	8.8	20	110
			184	780	38	19	40	16	15	160
			187	510	26	18	24	13	9.6	110
			190	1,500	98	36	70	26	42	80
			192	740	44	13	34	22	10	160
			195	780	19	14	23	5.8	14	88
			196	560	24	19	28	8.8	13	110
			198	500	19	15	17	8.0	15	92
			201	1,200	54	22	66	24	28	300
			204	1,700	41	26	29	11	24	140
			GM	847	35	20	34	13	17	123
			CL	620–1,160	24–50	16–25	24–46	8.8–18	12–24	92–163
		Brain (8)	184	20	0.50	ND	0.33	ND	ND	0.70
			187	10	0.24	0.28	0.16	ND	ND	ND
			190	45	1.9	ND	1.0	ND	ND	2.7
			192	17	0.60	ND	0.40	ND	ND	2.0
			195	14	0.21	ND	0.48	0.14	0.13	2.4
			196	11	0.30	ND	0.33	ND	ND	1.2
			201	22	0.60	ND	0.60	ND	ND	1.8
			204	41	0.63	0.19	1.4	0.33	0.28	4.2
			GM	20	0.56	0.05	0.55	0.05	0.05	1.6
			CL	12–31	0.2–1.0	ND–0.2	0.3–0.9	ND–0.33	ND–0.28	0.7–3.0
		Muscle (10)	184	16	0.50	ND	0.63	0.28	ND	2.3
			184	11	0.40	ND	0.15	ND	ND	0.43
			187	5.3	0.22	ND	ND	ND	ND	ND
			190	38	1.9	ND	0.78	ND	ND	2.4
			192	10	0.30	ND	0.25	ND	ND	1.2
			195	7.5	0.21	ND	0.30	0.15	ND	0.78
			196	8.3	0.14	ND	0.19	ND	ND	0.78
			198	16	0.43	ND	0.15	ND	ND	0.70
			201	14	0.43	ND	0.38	ND	ND	1.2
			204	14	0.30	ND	0.48	0.25	ND	1.7
			GM	12	0.43	ND	0.31	0.06	ND	1.0
			CL	8.2–18	0.2–0.7		0.2–0.5	ND–0.2		0.5–1.6
Bottlenose dolphin	F	Blubber (2)	211	1,600	100	46	30	11	9.6	420
			255	2,500	130	54	ND	11	ND	450
		Muscle (2)	211	49	3.7	0.11	1.7	0.53	ND	15
			255	18	0.70	ND	ND	ND	ND	5.5
Pilot whale	F	Blubber (1)	457	30	1.1	1.6	1.6	ND	0.78	8.8
		Muscle (1)	457	0.53	ND	ND	ND	ND	ND	ND
	M	Blubber (1)	670	110	3.4	6.4	4.8	1.1	4.4	14
		Muscle (1)	670	6.5	0.30	0.14	0.24	0.18	0.10	0.88
Harbor porpoise	F	Blubber (1)	140	270	36	11	13	2.2	2.6	84
		Brain (1)	140	2.2	0.15	ND	ND	ND	ND	0.65
		Muscle (1)	140	2.0	0.20	ND	ND	ND	ND	0.50
Dall's porpoise	F	Blubber (1)	183	190	16	14	14	5.2	6.6	94
		Muscle (1)	183	3.8	0.17	ND	ND	ND	ND	0.53
					HAWAII					
20°48′N, 156°28′W										
Rough-toothed dolphin	F	Blubber (4)	202	24	0.90	2.2	ND	0.60	1.1	38
			209	15	0.94	1.4	ND	0.86	2.0	26
			215	0.28	ND	ND	ND	ND	ND	ND
			218	ND	ND	ND	ND	ND	ND	ND

TABLE 1. *Continued*

Locality and Species	Sex	Tissue (N)	TL	*p,p'*-DDE	*p,p'*-TDE	*p,p'*-DDT	*o,p'*-DDE	*o,p'*-TDE	*o,p'*-DDT	PCBs
		Brain (1)	218	ND	ND	ND	ND	ND	ND	ND
		Muscle (4)	202	0.21	ND	ND	ND	ND	ND	0.45
			209	0.12	ND	ND	ND	ND	ND	0.14
			215	ND	ND	ND	ND	ND	ND	ND
			218	ND	ND	ND	ND	ND	ND	ND
	M	Blubber (3)	211	7.6	0.62	0.70	ND	0.38	1.2	14
			228	4.2	ND	ND	ND	ND	0.32	7.0
			235	4.6	ND	ND	ND	ND	ND	7.2
		Brain (3)	211	ND	ND	ND	ND	ND	ND	ND
			228	0.78	ND	ND	ND	ND	ND	1.3
			235	0.48	ND	ND	ND	ND	ND	0.78
		Muscle (3)	211	ND	ND	ND	ND	ND	ND	ND
			228	0.22	ND	ND	ND	ND	ND	0.38
			235	0.25	ND	ND	ND	ND	ND	0.33
JAPAN										
34°15′N, 136°52′E										
Striped dolphin	F	Blubber (4)	210	0.64	ND	0.62	ND	ND	ND	1.8
			211	10	1.0	4.6	0.34	1.0	1.1	4.8
			223	8.2	1.4	3.2	0.28	0.55	1.1	4.0
			229	0.88	0.18	0.96	ND	0.14	ND	1.2
		Brain (4)	210	ND	ND	ND	ND	ND	ND	ND
			211	0.23	ND	ND	ND	ND	ND	ND
			223	0.18	ND	ND	ND	ND	ND	ND
			229	ND	ND	ND	ND	ND	ND	ND
		Muscle (4)	210	ND	ND	ND	ND	ND	ND	ND
			211	1.2	0.17	0.38	ND	ND	ND	0.63
			223	0.30	ND	ND	ND	ND	ND	0.85
			229	ND	ND	ND	ND	ND	ND	ND
	M	Blubber (1)	239	12	0.78	4.4	0.30	0.76	0.68	5.0
		Brain (1)	239	0.80	0.16	ND	ND	ND	ND	ND
		Muscle (1)	239	0.63	0.11	ND	ND	ND	ND	1.6
Pilot whale	F	Blubber (5)	332	3.8	0.78	1.6	0.48	0.36	0.24	32
			348	4.4	0.54	2.6	6.62	ND	0.28	6.2
			354	0.56	0.12	0.40	0.14	ND	ND	1.6
			356	1.9	0.32	1.3	0.62	0.12	0.28	0.60
			362	5.2	0.76	2.8	0.58	0.30	0.62	5.8
			GM	2.7	0.48	1.6	0.48	0.15	0.27	4.8
			CL	0.8–6.5	0.2–0.9	0.6–3.2	0.2–0.8	ND–0.4	0.03–0.57	0.4–24
	M	Blubber (1)	355	0.48	ND	0.40	0.12	ND	ND	2.0
34°30′N, 135°E										
Finless porpoise	F	Blubber (3)	118	42	42	26	16	3.4	2.4	88
			163	3.4	2.4	2.8	3.0	0.48	ND	18
			164	20	7.8	6.6	17.0	1.6	1.7	74
		Brain (1)	164	0.15	ND	ND	ND	ND	ND	1.8
		Muscle (2)	164	ND	ND	ND	ND	ND	ND	0.43
			163	0.16	ND	ND	ND	ND	ND	0.70
	M	Blubber (1)	167	62	30	15	15.6	5.8	3.6	96
		Muscle (1)	167	0.45	0.20	ND	ND	ND	ND	1.1
34°30′N, 137°E										
Finless porpoise	F	Blubber (1)	124	14	7.2	7.8	5.6	1.0	1.0	50
		Muscle (1)	124	0.10	ND	0.14	ND	ND	ND	0.63
	M	Blubber (1)	148	32	19	17	10.0	3.4	2.8	64
		Brain (1)	148	0.26	ND	ND	ND	ND	ND	1.3
		Muscle (1)	148	0.35	ND	ND	ND	ND	ND	1.2
35°30′N, 139°50′E										
Common dolphin	F	Blubber (1)	183	0.54	0.46	0.36	0.12	0.34	ND	2.4
		Muscle (1)	183	ND	ND	ND	ND	ND	ND	0.30
	M	Blubber (1)	144	8.2	1.8	4.0	0.40	1.8	0.72	6.4
		Muscle (1)	144	0.28	ND	ND	ND	ND	ND	0.35
43°35′N, 145°55′E										
Dall's porpoise	F	Blubber (1)	184	3.0	1.1	1.4	1.0	1.3	0.78	3.4
		Brain (1)	184	ND	ND	ND	ND	ND	ND	ND
		Muscle (1)	184	ND	ND	ND	ND	ND	ND	0.16

NOTE: (N) = number of individuals sampled; TL = total length of animal in cm; F, M = female, male; ND = not detected; GM = geometric mean; CL = 95% confidence limits.

TABLE 2. *Organochlorine residues other than ΣDDT and PCBs in blubber of small cetaceans from the Pacific and South Atlantic Oceans, November 1968–June 1976*[1]

Locality and Species	Sex (N)	TL	Dieldrin	HCB	Toxaphene	Heptachlor epoxide	*trans*-Nonachlor	Oxychlordane
URUGUAY								
24°22′S, 53°46′W								
Franciscana dolphin	F (3)	116	0.22	0.10	ND	ND	0.12	ND
		133	0.20	ND	ND	ND	ND	ND
		138	0.20	0.10	ND	ND	0.10	ND
	M (5)	114	1.0	ND	ND	ND	0.20	ND
		116	0.52	0.18	ND	ND	0.24	ND
		130	0.38	ND	ND	ND	0.10	ND
		140	0.40	0.12	ND	ND	0.18	ND
		145	0.30	0.10	ND	ND	0.20	ND
		GM	0.50	0.08	ND	ND	0.18	ND
		CL	0.2–0.9	ND–0.2			0.1–0.25	
EASTERN TROPICAL PACIFIC								
3°26′S, 91°41′W								
Fraser's dolphin	M (1)	168	ND	ND	ND	ND	ND	ND
4°50′S, 88°12′W								
Striped dolphin	F (2)	186	0.14	ND	ND	ND	ND	ND
		187	0.14	ND	ND	ND	0.18	ND
	M (1)	195	0.20	ND	ND	ND	ND	ND
7°0′N, 86°30′W								
Striped dolphin	F (1)	192	ND	ND	ND	ND	ND	ND
7°46′N, 84°20′W								
Striped dolphin	F (1)	131	ND	ND	ND	ND	0.22	ND
9°41′N, 98°7′W								
Striped dolphin	M (2)	148	0.84	ND	5.6	0.30	ND	ND
		160	ND	ND	4.0	0.26	ND	ND
17°53′N, 105°6′W								
Striped dolphin	F (1)	180	0.44	ND	ND	ND	0.48	ND
18°25′N, 110°48′W								
Striped dolphin	F (1)	194	ND	ND	ND	ND	ND	ND
20°6′N, 110°29′W								
Striped dolphin	F (4)	136	0.30	ND	ND	ND	0.16	ND
		178	0.40	ND	ND	ND	0.64	ND
		188	ND	ND	ND	ND	ND	ND
		190	0.28	ND	ND	ND	0.24	ND
CALIFORNIA								
32°33′N–35°12′N								
Common dolphin	F (3)	182	ND	0.13	ND	ND	ND	ND
		192	1.1	ND	ND	0.34	ND	0.12
		210	ND	0.24	ND	ND	ND	ND
	M (10)	184	ND	0.18	ND	ND	ND	ND
		184	1.1	0.10	ND	0.30	ND	0.20
		187	ND	0.12	ND	ND	ND	ND
		190	ND	0.34	ND	2.0	ND	1.2
		192	1.4	0.14	ND	ND	10	0.50
		195	ND	0.14	ND	ND	ND	ND
		196	ND	ND	ND	0.12	0.82	0.32
		198	ND	ND	ND	ND	ND	0.20
		201	ND	0.10	ND	0.22	ND	0.20
		204	ND	0.24	ND	ND	ND	ND
		GM	0.18	0.13	ND	0.18	0.35	0.22
		CL	ND–0.5	0.1–0.2		ND–0.5	ND–1.3	0.02–0.5
Bottlenose dolphin	F (2)	211	ND	0.34	ND	0.62	14	3.4
		255	6.4	0.46	ND	ND	21	4.0
Pilot whale	F (1)	457	ND	ND	ND	ND	ND	ND
	M (1)	670	ND	ND	ND	ND	ND	ND
Harbor porpoise	F (1)	140	1.0	0.18	ND	0.18	5.0	ND
Dall's porpoise	F (1)	183	0.40	0.28	ND	ND	0.56	ND

TABLE 2. *Continued*

Locality and Species	Sex (N)	TL	Dieldrin	HCB	Toxaphene	Heptachlor epoxide	*trans*-Nonachlor	Oxychlordane
HAWAII								
20°48′N, 156°28′W								
Rough-toothed dolphin	F (4)	202	0.32	ND	ND	ND	0.64	ND
		209	0.66	ND	ND	ND	1.0	ND
		215	ND	ND	ND	ND	ND	ND
		218	ND	ND	ND	ND	ND	ND
	M (3)	211	0.36	ND	ND	ND	0.50	ND
		228	ND	ND	ND	ND	ND	ND
		235	ND	ND	ND	ND	ND	ND
JAPAN								
34°15′N, 136°52′E								
Striped dolphin	F (4)	210	ND	ND	ND	ND	ND	ND
		211	0.28	0.08	0.18	ND	ND	ND
		223	0.34	ND	ND	ND	0.24	ND
		229	ND	ND	0.12	ND	ND	ND
	M (1)	239	ND	0.10	0.18	ND	0.12	ND
Pilot whale	F (5)	332	ND	ND	0.14	ND	ND	ND
		348	ND	0.06	ND	ND	ND	ND
		354	ND	ND	ND	ND	ND	ND
		356	ND	ND	0.14	ND	ND	ND
		362	1.2	ND	0.14	ND	ND	ND
		GM	0.17	0.01	0.08	ND	ND	ND
		CL	ND–0.8	ND–0.05	ND–0.2			
	M (1)	355	ND	ND	0.12	ND	ND	ND
34°30′N, 135°E								
Finless porpoise	F (3)	118	16	0.64	ND	ND	ND	ND
		163	2	ND	ND	ND	ND	ND
		164	12	0.38	0.22	ND	ND	0.36
	M (1)	167	32	0.42	0.16	0.28	1.1	0.12
34°30′N, 137°E								
Finless porpoise	F (1)	124	16	0.22	ND	0.34	0.40	ND
	M (1)	148	38	0.34	0.14	0.82	ND	0.46
35°30′N, 139°50′E								
Common dolphin	F (1)	183	0.14	0.06	0.14	0.04	ND	ND
	M (1)	144	0.62	0.26	0.36	ND	0.46	ND
43°35′N, 145°55′E								
Dall's porpoise	F (1)	184	0.40	0.28	ND	ND	0.56	ND

NOTE: (N) = number of individuals sampled; TL = total length of animal in cm; F, M = female, male; ND = not detected; GM = geometric mean; CL = 95% confidence limits.

[1] Brain and muscle tissues (sample sizes identical to those in Table 1) had no detectable residues of the compounds listed above except as noted in the text.

in blubber of the male common dolphin from Tokyo Bay and at 0.56 ppm in blubber of a male finless porpoise captured at sea near Himeji, Hyogo Prefecture, Japan. Concentrations of *cis*-nonachlor were 2.2 ppm and 10 ppm in blubber of two beach-stranded southern California female bottlenose dolphins. Residues of *cis*-chlordane were positively identified in blubber of the following cetaceans: two male Franciscana (*Pontoporia blainvillei*) from the western South Atlantic off Uruguay (0.94 ppm and 0.12 ppm); a female striped dolphin collected at 20° 6′N, 110° 29′W (0.18 ppm); a female bottlenose dolphin (13 ppm), a female common dolphin (6.4 ppm), and six male common dolphins (geometric mean 9.8 ppm, range 5.8–36 ppm) from southern California.

RESIDUES BY TISSUES

Blubber has a high lipid content, and organochlorine concentrations are therefore consistently higher in this tissue than in brain or muscle. However, statistical comparisons of residue concentrations within locality, species, and sex are possible only for tissues of male common dolphins from southern California. Mean concentrations of *p,p′*-DDE in blubber, brain, and muscle of these animals (Table 1) differed significantly ($P < 0.01$; two-way analysis of variance on ln + 1 transformed data, mean separation by Student-Newman-Keuls test). Mean PCB concentrations in blubber were significantly greater than those in brain or muscle ($P < 0.01$). Brain and muscle PCB concentrations did not differ ($P > 0.05$). Residue levels of DDE in these three tissues were significantly correlated in common dolphins from southern California and in striped dolphins from the Eastern Tropical Pacific (Table 3). PCB residues in common dolphins showed significant correlation only between brain and muscle (Table 3).

TABLE 3. *Simple linear correlation coefficients (r) between blubber, brain, and muscle tissues containing residues of DDE and PCBs in 13 common dolphins collected at southern California beaches and of DDE in 14 striped dolphins from the Eastern Tropical Pacific, November 1968–June 1976*

Species	Residue		Blubber/ Brain	Blubber/ Muscle	Brain/ Muscle
Common dolphins	DDE	*r*	0.92**	0.65*	0.83**
		DF	7	11	7
	PCB	*r*	−0.04	0.09	0.75**
		DF	7	11	7
Striped dolphins	DDE	*r*	0.99**	0.97**	0.99**
		DF	9	12	9

* $P < 0.05$.
** $P < 0.01$.

PCBs were not detected in muscle and occurred in only one brain sample of Eastern Tropical Pacific striped dolphins.

Except in heavily contaminated individuals, the principal organochlorine compounds found in blubber were not detected in brain or muscle at the same frequencies (Table 1). For example, DDE did not appear in brains of Eastern Tropical Pacific striped dolphins except when blubber residues exceeded about 5–10 ppm (Fig. 1). In contrast, all southern California common dolphins had detectable DDE residues in brains, but blubber residues were 360 ppm or greater (Fig. 1). Similarly, only two of the compounds listed in Table 2 that were found in blubber were also found in brain or muscle. Dieldrin occurred at 0.20 ppm in the muscle of a male finless porpoise collected off Himeji, Japan. Brain or muscle tissues of three common dolphins from southern California beaches contained residues of *trans*-nonachlor. The brain of one female contained 0.25 ppm and those of two males contained 0.33 ppm and 0.23 ppm. Muscle of the two males contained *trans*-nonachlor at 0.16 ppm and 0.12 ppm.

RESIDUES BY LOCALITY

Uruguay—Five male and three female Franciscana were collected offshore from Punta del Diablo, Dept. Rocha (34° 22′S, 53° 46′W). Dieldrin, PCBs, and DDT and its metabolities were present in blubber of all individuals (Tables 1 and 2). Other organochlorines present in blubber included HCB, *trans*-nonachlor, and *cis*-chlordane. PCBs were the only organochlorines detected in muscle and brain. This was the only locality in which residues of *p,p′*-DDT exceeded those of *p,p′*-DDE in blubber (Table 4).

Eastern Tropical Pacific—Striped dolphins were collected at sea at latitudes ranging from 4° 50′S to 20° 6′N (Table 1). In general, striped dolphins from these localities had relatively low to moderate blubber concentrations of PCBs and DDT-related compounds (Table 1). Exceptions included two young males collected at 9° 41′N, 98° 7′W, which contained an

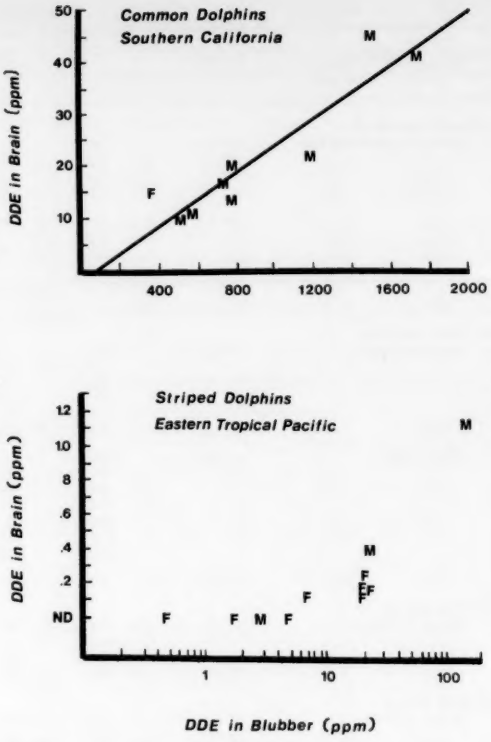

FIGURE 1. *Concentrations of DDE in brains of Southern California common dolphins and Eastern Tropical Pacific striped dolphins in relation to DDE concentrations in blubber (M = male, F = female). Line depicted in top portion of figure is described by equation* $Y = -1.28 + 0.025X$.

average of 115 ppm *p,p′*-DDE in blubber as well as residues of PCBs, dieldrin, and heptachlor epoxide (Tables 1 and 2); toxaphene residues in these specimens were the highest reported in this study. Few compounds other than PCBs and those related to DDT were found in blubber of other striped dolphins. PCBs were not detected in the two females taken at 4° 50′S, 88° 12′W but were found in the male. With the exception of *o,p′*-TDE in muscle tissues of the two males from 9° 41′N, 98° 7′W, brain and muscle tissues of Eastern Tropical Pacific striped dolphins did not possess detectable residues of any organochlorines other than *p,p′*-DDE and *p,p′*-TDE.

The blubber of one male Fraser's dolphin collected at 3° 26′S, 91° 41′W contained residues of PCBs and DDT-related compounds. The predominant compound was *p,p′*-DDE at 7.2 ppm (Table 1).

California—Five species of small cetaceans were sampled on or near southern California beaches at latitudes

TABLE 4. *Ratios of* p,p'-*DDT to* p,p'-*DDE in blubber of small cetaceans grouped according to oceanic region or the nearest major land masses at the latitudes of collection, November 1968–June 1976*

Species	Sample Size	$\frac{p,p'\text{-DDT}}{p,p'\text{-DDE}} \pm$ Std Dev
SOUTHERN CALIFORNIA		
Common dolphins	(13)	0.03 ± 0.01
Bottlenose dophins	(2)	0.03 ± 0.004
Pilot whales	(2)	0.06 ± 0.004
Harbor porpoise	(1)	0.04
Dall's porpoise	(1)	0.07
HAWAII		
Rough-toothed dolphins	(6)	0.05 ± 0.05
EASTERN TROPICAL PACIFIC		
Fraser's dolphin	(1)	0.25
Striped dolphins	(14)	0.34 ± 0.18
JAPAN		
Striped dolphins	(5)	0.66 ± 0.35
Pilot whales	(6)	0.63 ± 0.14
Finless porpoise	(6)	0.52 ± 0.21
Common dolphins	(2)	0.58 ± 0.13
Dall's porpoise	(1)	0.47
URUGUAY		
Franciscana	(8)	1.97 ± 0.90

32° 33′N and 35° 12′N. These animals were heavily contaminated with organochlorine residues, including particularly high concentrations of metabolites and isomers of DDT. The inshore-feeding coastal bottlenose dolphins were the most heavily contaminated. Blubber from two immature females stranded in San Diego County in July and August 1976 contained 2,695 and 1,797 ppm ΣDDT, over 400 ppm PCBs, and lesser quantities of dieldrin, heptachlor epoxide, HCB, oxychlordane, *trans*-nonachlor, and *cis*-chlordane (Tables 1 and 2). Blubber of comon dolphins had concentrations of up to 1,831 ppm ΣDDT and 300 ppm PCBs. Up to 48 ppm ΣDDT occurred in brains. With the exception of dieldrin, HCB, and toxaphene, residues of all organochlorines listed in Tables 1 and 2 were higher in these two species in southern California than in any species at any other locality.

High concentrations of organochlorines were also present in the other California specimens. Residues of ΣDDT in blubber reached 335 ppm in an immature female harbor porpoise (*Phocoena phocoena*) stranded in San Luis Obispo County and 246 ppm in an adult female Dall's porpoise (*Phocoenoides dalli*) stranded in San Diego County. Lowest ΣDDT residues in blubber were 35 ppm in an immature female pilot whale incidentally caught off Los Angeles County.

Hawaii—Tissue samples were analyzed from seven rough-toothed dolphins (*Steno bredanensis*) that mass-stranded at Kihei Beach, Maui (20° 58′N, 156° 28′ W). One adult female did not possess detectable residues of organochlorine compounds. This was the sole individual in the entire study that was free of contamination. Isomers and metabolites of DDT were present in blubber of other individuals at relatively low concentrations. PCBs reached 38 ppm in one female. Dieldrin and *trans*-nonachlor were the only additional organochlorines detected at this locality. The blubber of a 93-cm near-term fetus (not tabulated) contained one compound, *p,p′*-DDE, at 0.54 ppm. Maternal blubber also contained only *p,p′*-DDE, at a concentration of 0.28 ppm.

Japan—Striped dolphins and pilot whales were caught by fishermen at Taiji, Wakayama Prefecture (34° 15′N, 136° 52′E), and finless porpoises were taken at Ise Bay (34° 30′N, 137° E) and the Seto Inland Sea (34° 30′N, 135° E). These specimens contained a wide range of organochlorine residues (Tables 1 and 2), including the highest blubber concentrations of dieldrin and HCB found in the study (up to 38 ppm and 0.64 ppm, respectively, in finless porpoises). Toxaphene or toxaphene-like compounds were found in tissues of individuals of all three species and in common dolphins stranded in Tokyo Bay (35° 30′N, 139° 50′E). Toxaphene was detected elsewhere only in striped dolphins taken at 9° 41′N, 98° 7′W. As previously noted, the two common dolphins from Tokyo Bay contained mirex and endrin in addition to the compounds listed in Tables 1 and 2. The highest concentrations of *p,p′*-DDE and PCBs among Japanese cetaceans occurred in finless porpoises, which are also the most highly restricted to inshore feeding. Concentrations of PCBs were higher in blubber of Japanese finless porpoises than in cetaceans from all other localities except southern California. Ratios of *p,p′*-DDT to *p,p′*-DDE were higher in Japanese cetaceans than in those from all other localities except the western South Atlantic off Uruguay (Table 4).

Discussion

Small cetaceans may generally be expected to reflect the extent of local organochlorine contamination in marine ecosystems: they occupy high trophic levels, are large nonmigratory, and are relatively long-lived (*21*). This premise is dramatically supported by residue concentrations in animals from southern California waters. The oceanic input of PCBs from five southern California sewage treatment plants was estimated at nearly 20,000 kg per year in 1972 (*20*), and one Los Angeles area pesticide manufacturer was responsible for a continuous discharge of DDT at sewer outfalls for over 20 years (*19*). Shortly before cessation of this latter practice in 1970, the input of ΣDDT (80 percent of which was DDT) to the ocean from this source alone was estimated to be 250 kg per day (*19*). Cetaceans collected in this region during our study show heavy

contamination with these compounds. With the exception of a single immature female pilot whale taken at sea, blubber from all specimens from beaches or waters off southern California contained residues of *p,p'*-DDE in excess of 100 ppm. Mean concentrations of this compound in blubber of male common dolphins were more than seven times greater than the highest individual values in any species from any other locality in the present study. Maximum residues of *p,p'*-DDE in blubber of two immature female coastal bottlenose dolphins were even higher; *p,p'*-DDE residues were more than twice the highest previously published concentrations of this compound in cetaceans (*23*). PCB residues in blubber of the California *Tursiops* also exceeded previously published concentrations for cetaceans from all parts of the world yet investigated except perhaps the Mediterranean coast of France (*1, 25*). Blubber residues of ΣDDT in one of these females reached 2,695 ppm, a value equivalent to the 2,678 ppm reported in an adult male California sea lion (*Zalophus californianus*) collected near Año Nuevo Island, California (37° 5'N, 122° 20'W), in 1970 (*18*). Concentrations of ΣDDT in California pinnipeds and cetaceans are to the authors' knowledge the highest known for any populations of wild mammals.

Cetaceans from other localities had lower concentrations of the principal organochlorine residues (DDT and PCBs), although these compounds were detected in all species at all localities. The use of DDT on neighboring land masses has presumably had varying histories. Direct discharge of DDT at southern California outfalls took place for over 20 years and had been curtailed for several years before specimen collection (*19*). In addition, the general use of DDT in the United States ended in 1972. Mean ratios of *p,p'*-DDT to *p,p'*-DDE are very low, ranging from 0.03 to 0.07 ppm in all species collected in waters off Hawaii and California (Table 4); long-term accumulation of the persistent metabolite DDE in marine ecosystems, as well as a lack of recent input of DDT, contribute to these low ratios. Mean ratios in cetaceans from other localities (Table 4) are at least an order of magnitude higher, ranging from 0.25 ppm to 0.66 ppm in Eastern Tropical Pacific and Japanese waters to the extreme in Franciscana dolphins from the Western South Atlantic, where residues of DDT exceed those of DDE. (Decomposition of DDT during storage of specimens can also influence these ratios (*7*). However, in comparison to geographic effects, this loss appears to have contributed little to the contrasting values seen in the present study: tissues of California cetaceans, for example, were stored for 23.5 ± 4 months whereas Uruguayan specimens were held 23.9 ± 0.3 months before they were analyzed.) Franciscana are very restricted to coastal waters of this region (*4*; R. L. Brownell, Jr., personal observations), and also extensively use the estuary of the Rio de la Plata. Widespread deforestation and agricultural cultivation are recent phenomena in much of the watershed draining into the Rio de la Plata system, particularly in southeastern Brazil (*17*). In addition, sharp increases in the use of DDT (and loss of fish and wildlife) have recently occurred near Brazil's extensive Lagoa dos Patos (*10*). The entrance to this lagoon is about 250 km north of the Uruguay collection site and the Brazilian Current in this area moves south along the coast. The introduction of DDT into the areas inhabited by Franciscana dolphins thus appears to be relatively recent and ongoing.

Cetaceans from Japanese waters contained a wide array of organochlorine compounds. This is particularly true of finless porpoises, which are the most restricted inshore feeders of all cetaceans surveyed in Japan. Although we know of no accounts documenting the degree of organochlorine contamination in other components of marine ecosystems at these localities, areas such as Ise Bay and the Seto Inland Sea possess some of the most densely industrialized sections of coastline in the world (*22*). This industrialization, along with intensive agricultural practices, apparently correlates with the diversity of organochlorine compounds seen in Japanese cetaceans. Eastern Tropical Pacific striped dolphins usually contained fewer kinds of residues in comparison with Japanese striped dolphins, but concentrations of DDT and PCBs were generally higher. Sources of contamination in these tropical seas are unknown.

In addition to *o,p'*-isomers of DDT and its metabolites, a number of other compounds, which have not been commonly reported in earlier studies, were detected in blubber of cetaceans (Table 2). This is the first report of endrin, toxaphene, *cis*-nonachlor, oxychlordane, or *trans*-nonachlor in tissues of cetaceans. Maximum residues of dieldrin (38 ppm in blubber of Japanese finless porpoises) were more than twice the highest previously reported concentrations (*15*). Residues of *cis*-chlordane in blubber of California common dolphins were nearly three times greater than those in previous reports (*23*). Other than our report of mirex in Japanese cetaceans, the only other marine mammals in which this compound has been detected are harbor seals from the Netherlands (*24*).

Although the present study has shown heavy and widespread contamination of small cetaceans with organochlorine compounds, the impact of this exposure has not yet been determined. Concentrations of ΣDDT and PCBs in blubber lower than those reported here have been associated with impaired reproduction in pinnipeds, but cause-and-effect relationships are not well defined. Population declines and uterine pathology

have been documented in Baltic Sea ringed seals (*Pusa hispida*); nonpregnant females with abnormal uteri had significantly higher mean blubber concentrations of ΣDDT and PCBs (130 ppm and 110 ppm) than pregnant females (88 ppm and 73 ppm) (*13*). On this basis it was concluded that organochlorines were responsible for the observed effects (*13*). However, because loss of organochlorines through lactation and pregnancy is an avenue unavailable to female mammals with imparied reproduction, higher residue concentrations may be expected to occur in non-reproducing females regardless of cause of reproductive failure. Residues in nonpregnant ringed seals with normal uteri, for example, did not differ significantly from those in nonpregnant females exhibiting the pathological changes [$P < 0.05$, *t*-test calculated from data of Helle et al. (*13*)]. A more marked association has been noted between high blubber residues of both ΣDDT (range 626–1039 ppm) and PCBs (85–145 ppm) and premature pupping in California sea lions (*6*). Females producing full-term pups had considerably lower concentrations of these compounds (51–203 ppm ΣDDT, 12–25 ppm PCB) (*6*). Although this relationship appears stronger than that implied by the ringed seal data, it is confounded by additional factors: premature and full-term parturient females differed in age and probably used different feeding areas, and abortion-inducing diseases were present in the population (*8*). It is currently hypothesized that interactions between organochlorines and some of these other factors are responsible for premature pupping (*8*).

Despite the equivocal nature of field observations of associations between high concentrations of organochlorines in blubber and reproduction in pinnipeds, toxic effects of PCBs and DDT or its metabolites, including severe reproductive impairment, have been experimentally verified in other mammals and birds (*3, 11, 12*). Drastic population reductions due to DDE exposure have been well documented in fish-eating birds from marine waters off southern California (*2, 9, 16*). Although the impact of organochlorine pollutants on small cetaceans has not yet been determined, the severe degree of contamination found in cetaceans from these same waters, due largely to what would presently be considered industrial negligence (*19*), should in itself be cause for serious concern.

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Use of Selected Freshwater Bivalves for Monitoring Organochlorine Pesticide Residues in Major Mississippi Stream Systems, 1972–73

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ABSTRACT

Seven species of freshwater Pelecypoda, Amblema costata, Corbicula manilensis, Elliptio crassidens, Lampsilis anadontoides, Lampsilis claibornensis, Megalonaias gigantea, *and* Plectomerus dombeyanus, *were collected and monitored for pesticide content during 1972 and 1973. Thirteen collection sites, representing five major river basins in the state of Mississippi, were sampled and compared. During the 24-month study, 26 water samples and 58 claim samples from the five river basins were analyzed. Individual samples weighed from 8 g to 20 g and consisted of 1–30 clams, depending on size. Residues of toxaphene and methyl parathion were found only in 1973 water samples. The study shows that freshwater clams are effective monitors of pesticide content. The tendency of clams to concentrate pesticides and their corresponding ability to eliminate them varies with species. Significant reductions in DDT and a corresponding buildup of p,p'-TDE were noted in 1973, following the limitations on the use of DDT and large-scale flooding throughout the state.*

Introduction

The tendency of Pelecypoda to accumulate high body levels of pesticides from water containing very small amounts has been demonstrated under laboratory conditions (*2, 5, 7*). In addition, Butler (*2*) and Fikes (*5*) have shown that bivalves are able to eliminate virtually all of their body burden of pesticides when placed in pesticide-free water. Other researchers have investigated pesticide accumulations by marine and freshwater species following periods of spraying or from known pesticides outfalls (*1, 3, 4, 6*).

The present study evaluates the tendency of clams to accumulate pesticides and their ability to eliminate them under field conditions. It was felt that a comparison of 1972 and 1973 samples should reflect purging of high body concentrations as a result of either or both of these factors: the banning in December 1972 of the widespread use of DDT, and the extensive flooding of the state of Mississippi during Spring 1973.

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Sampling

A survey of pesticide levels in selected species of freshwater clams in the major stream systems of the state of Mississippi was conducted in Summer 1972 in order to determine body concentrations of pesticides. All collections were made during periods of low stream flow (July through November). In 1973, clams were again collected from the same sites within respective river basins. Seven species of clams, *Amblema costata, Corbicula manilensis, Elliptio crassidens, Lampsilis anadontoides, Lampsilis claibornensis, Megalonaias gigantea,* and *Plectomerus dombeyanus,* were collected from the stream systems. Samples consisted of one to four species. Since species varied in size, sufficient numbers of individuals were collected to provide 8–20 g for extraction.

The 13 sampling sites are shown in Figure 1. Collections were made by hand from shallow water; in deeper areas, dip nets, dredges, and oyster tongs were used. Clams were refrigerated and returned to the laboratory in ice chests. Specimens were then identified, and their soft tissues were removed. Soft tissues were blotted on Whatman No. 1 filter paper to reduce excess water and were frozen at —15°C until extraction could be performed.

Analysis

Pesticides were extracted from clams by a modification of the procedure outlined in *Analysis of Pesticides in the Aquatic Environment* (*8*). Each sample was combined with three times its weight of sodium sulfate and extracted twice with equal portions of redistilled hexane by blending 3 minutes each time. The procedure gave 92 percent recovery for the first extraction and 8 percent recovery for the second.

The extracts were combined and filtered through Whatman No. 1 and No. 3 filter papers. The resulting extract was partitioned into acetonitrile and evaporated to dryness in a Kuderna-Danish concentrator over a steam bath. The residue was dissolved in 25 ml hexane

FIGURE 1. *Mississippi River Basin sampling sites, for monitoring pesticide residues in freshwater bivalves, 1972–73. Stations 1–3: Yazoo River Basin; Station 4: Big Black River Basin; Station 5: Tombigbee River Basin; Stations 6–8: Pearl River Basin; Stations 9–13: Pascagoula River Basin.*

and subjected to Florisil column cleanup: 4 inches Na_2SO_4 bottom layer, 10 g Florisil, 3 inches Na_2SO_4 top layer, ether–hexane (15 + 85) elution mixture.

Water samples were also taken in conjunction with the clam samples. They were collected in glass jars which were sealed with aluminum foil-lined screw caps. Samples were refrigerated in ice chests and extracted upon return to the laboratory, as follows: One liter of unfiltered water was extracted with a 100-ml solution of 85 percent hexane and 15 percent petroleum ether by rolling 1 hr on a concentric rotator. The resulting extract was eluted through approximately 6 inch anhydrous Na_2SO_4 in a size 224 Chromaflex (Kontes, 1 inch ID) column using a mixture of hexane–ethyl ether (85 + 15). The extract was concentrated to 5 ml in a Kuderna-Danish apparatus over a steam bath.

All samples were analyzed on a Micro Tek 220 gas chromatograph equipped with ^{63}Ni electron-capture detector. Instrument parameters and operating conditions follow:

Columns:	6 ft × ¼ inch OD glass column packed with a mixture of 1.5 percent OV–17 and 1.95 percent QF–1 (1972 and 1973) 10 percent DC–200 (1972 only) 3 percent OV–1 (1973 only) each on Chromosorb W (AW, DMCS, HP)
Temperatures:	injector 220°C column 185–195°C detector 260°C
Carrier gas:	prepurified nitrogen flowing at ca 60–80 ml/minute

In 1973, a 3 percent OV–1 column was substituted for the 10 percent DC–200 because of its greater sensitivity and lower retention time.

Residues were qualitatively identified by comparison with a standard. Quantitative measurements were based on peak height and expressed in μg/g (ppm) for clam samples and μg/liter (ppb) for water samples. Results were not corrected for recovery.

Results and Discussion

Sites from which clams containing the highest levels of pesticides were taken corresponded with areas of greatest agricultural usage. Highest detectable levels of toxaphene and DDT and its metabolites were found in clams from sampling sites within the Yazoo, Big Black, and Tombigbee River Basins (Fig. 1). Watersheds of these basins are within the most highly exploited agricultural areas of Mississippi (Statistical Reporting Service, U.S. Department of Agriculture, 1973, personal communication). Pesticide residues were also detected in clams from the northern areas of the Pearl River Basin which drains farmlands. Clams from the Pascagoula River Basin contained the lowest levels of pesticides (Table 1).

Pesticide residues were not found in water samples collected during 1972. Residues of toxaphene and/or methyl parathion were detected in water samples collected during 1973 from sites within the Yazoo, Big Black, and Tombigbee River Basins (Table 2). Detectable levels of toxaphene and methyl parathion in water samples taken in 1973 reflect in part the increased use of these pesticides, following the limits imposed on use of DDT. The extreme insolubility of DDT in water may have precluded its detection in trace amounts in the 1972 samples.

Overall pesticide residues in clams from sites within the Yazoo River Basin were less in 1973 than in 1972 (Table 1). With respect to DDT, there were large-scale reductions of both *o,p'*-DDT and *p,p'*-DDT in clams. Residues of *p,p'*-TDE, a first-order breakdown product of DDT, remained constant or changed only slightly in 1973. Increases of *p,p'*-TDE coincided with subsequent decreases in *p,p'*-DDT levels. Residues of *p,p'*-DDE declined slightly or remained constant. Decrease in DDT, an increase in TDE, and an overall elimination of pesticides indicates that the clams may be metabolizing the DDT. This purging would have been accomplished when the entrance of DDT into the streams was reduced. Limited spraying or increased dilution would have caused such a reduction.

A second and possibly better explanation for the variations in observed levels of DDT and its metabolites would be runoff. It is well known that runoff is the strongest contributor of pesticides to streams which drain farmland. During the present two-year study, clams apparently assimilated greater amounts of TDE and lesser amounts of DDT as the levels entering the streams changed.

Flooding of the Mississippi River during Spring 1973, as well as limitations on DDT use, probably influenced these fluctuations of residues in clams throughout the Yazoo River Basin. Sampling sites within this basin are located near the area flooded and also drain more farmland than do any of the previously mentioned basins.

Toxaphene residues in clams were lower at most sites in 1973; however, residues in water increased. Water samples were taken near the surface, and increases in residues are probably the result of local spraying rather than runoff.

Reductions of all metabolites of DDT were observed at the sampling site within the Big Black River Basin. Levels of *p,p'*-TDE decreased, from 0.36 ppm to 0.09 ppm. Decrease in *p,p'*-DDT levels were even greater, from 0.67 ppm to 0.06 ppm; therefore, the same pattern was followed. The single sampling site from this

TABLE 1. *Comparison of 1972 and 1973 organochlorine residues in bivalves from major stream systems of the state of Mississippi*

	Residues µg/g															
	Chlordane		Endrin		Toxaphene		*p,p'*-DDE		*p,p'*-TDE		*p,p'*-DDT		*o,p'*-DDT		ΣDDT	
Yazoo Basin:	1972	1973	1972	1973	1972	1973	1972	1973	1972	1973	1972	1973	1972	1973	1972	1973
COLDWATER RIVER (QUITMAN COUNTY—R10W, T28N, 35)																
Corbicula manilensis	ND	ND	ND	ND	2.87	1.39	0.25	0.10	0.15	0.14	0.54	0.05	0.13	ND	1.07	0.28
Lampsilis anadontoides	ND	ND	ND	ND	TR[1]	0.48	0.08	0.04	0.06	0.04	0.11	0.02	ND	ND	0.25	0.10
SUNFLOWER RIVER (SUNFLOWER COUNTY—R4W, T18N, 8)																
Amblema costata	ND	ND	ND	ND	4.02	3.65	0.21	0.24	0.26	0.33	0.11	ND	0.53	0.22	1.11	179
Lampsilis anadontoides	ND	ND	ND	ND	4.86	1.51	0.28	0.12	0.27	0.16	0.22	ND	1.00	0.05	1.78	0.31
Plectomerus dombeyanus	ND	ND	ND	ND	1.01	0.75	0.05	0.10	0.10	0.14	ND	ND	0.08	0.03	0.23	0.28
STEEL BAYOU (ISSAQUENA COUNTY—R8W, T10N, 12)																
Amblema costata	ND	ND	ND	ND	1.09	0.72	0.12	0.18	0.13	0.16	ND	ND	0.07	0.01	0.32	0.26
Corbicula manilensis	ND	ND	ND	ND	7.68	3.62	0.39	0.23	0.40	0.49	ND	ND	0.53	0.10	1.32	0.81
Lampsilis anadontoides	ND	ND	ND	ND	1.93	1.11	0.18	0.11	0.21	0.21	ND	ND	0.12	0.04	0.51	0.35
Plectomerus dombeyanus	ND	ND	ND	ND	0.84	0.62	0.08	0.08	0.10	0.14	ND	ND	0.07	0.04	0.25	0.25
Big Black River Basin:																
BIG BLACK RIVER (WARREN COUNTY—R5W, T6N, 22)																
Corbicula manilensis	ND	ND	ND	ND	4.43	3.78	0.27	0.07	0.36	0.09	0.10	ND	0.67	0.06	1.40	0.22
Lampsilis anadontoides	ND	ND	ND	ND	TR[1]	1.01	0.04	0.02	0.05	0.03	ND	ND	0.06	0.02	0.14	0.06
Tombigee River Basin:																
TOMBIGBEE RIVER (LOWNDES COUNTY—R18W, T18S, 17)																
Megalonaias gigantea	ND	ND	ND	TR[1]	0.92	0.22	0.08	0.04	0.06	0.02	0.08	0.03	ND	ND	0.22	0.10
Pearl River Basin:																
PEARL RIVER (HINDS COUNTY—R1E, T6N, 36)																
Amblema costata	0.01	ND	ND	ND	ND	0.13	0.05	TR[2]	0.02	TR[2]	TR[2]	TR[2]	ND	ND	0.07	TR[2]
Lampsilis anadontoides	0.01	ND	ND	ND	ND	TR[1]	0.03	TR[2]	TR[2]	TR[2]	ND	ND	ND	ND	0.03	TR[2]
Plectomerus dombeyanus	TR[2]	ND	ND	ND	ND	0.13	0.02	0.01	TR[2]	TR[2]	ND	ND	ND	ND	0.02	0.01
Corbicula manilensis	ND	ND	ND	ND	ND	TR[1]	0.01	TR[2]	ND	TR[2]	ND	ND	ND	ND	0.01	TR[2]
Lampsilis anadontoides	ND	ND	ND	ND	ND	TR[1]	ND	TR[2]	ND	TR[2]	ND	ND	ND	ND	ND	TR[2]
PEARL RIVER (PEARL RIVER COUNTY—R18W, T5S, 29)																
Corbicula manilensis	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND
Pascagoula River Basin:																
CHUNKY RIVER (NEWTON COUNTY—R13E, T6N, 36)																
Corbicula manilensis	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND
Elliptio crassidens	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND
Lampsilis anadontoides	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND
CHICKASAWHAY RIVER (WAYNE COUNTY—R7W, T8N, 10)																
Corbicula manilensis	ND	ND	ND	ND	ND	TR[1]	ND	TR[2]	ND	TR[2]	ND	TR[2]	ND	ND	ND	TR[2]
Elliptio crassidens	ND	ND	ND	ND	ND	0.30	ND	TR[2]	ND	TR[2]	ND	0.03	ND	ND	ND	0.03
LEAF RIVER (JONES COUNTY—R13W, T6N, 33)																
Corbicula manilensis	ND	ND	ND	ND	ND	TR[1]	0.04	0.02	0.05	0.01	0.05	0.02	ND	ND	0.13	0.05
Lampsilis anadontoides	ND	ND	ND	ND	ND	TR[1]	0.01	0.01	TR[2]	TR[2]	TR[2]	TR[2]	ND	ND	0.01	0.01
PASCAGOULA RIVER (GEORGE COUNTY—R8W, T2S, 23)																
Corbicula manilensis	ND	ND	ND	ND	ND	ND	TR[2]	TR[2]	TR[2]	TR[2]	ND	ND	ND	ND	TR[2]	TR[2]
Lampsilis anadontoides	ND	ND	ND	ND	ND	ND	ND	TR[2]	ND	TR[2]	ND	TR[2]	ND	ND	ND	TR[2]
Plectomerus dombeyanus	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND
BLACK CREEK (GEORGE COUNTY—R8W, T3S, 14)																
Lampsilis claibornensis	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND

NOTE: ND = None detected.
[1] Less than 0.1 µg/g for toxaphene.
[2] Less than 0.01 µg/g for DDT and metabolites.

TABLE 2. *Pesticide residues in water samples taken concurrently with Pelecypoda samples from major stream systems in the state of Mississippi, 1973*

Sampling site	Organochlorines	Residues, µg/liter (ppb)
Yazoo River Basin:		
Coldwater River (Quitman County—R10W, T28N, 35)	Toxaphene	0.46
	p,p'-DDE	0.01
Sunflower River (Sunflower County—R4W, T18N, 8)	Toxaphene	0.44
Steele Bayou (Issaquena County—R8W, T10N, 12)	Toxaphene	2.11
	Methyl parathion	0.25
Big Black River Basin:		
Big Black River (Warren County—R5W, T6N, 22)	Toxaphene	1.42
	Methyl parathion	0.46
Tombigbee River Basin:		
Tombigbee River (Lowndes County—R18W, T18S, 17)	Methyl parathion	0.08
Pearl River Basin:		
Three sampling sites	None detected	
Pascagoula River Basin:		
Five sampling sites	None detected	

basin is the lower end of the more highly agricultural, or Delta, region of Mississippi.

The single species collected from the Tombigbee River Basin, *M. gigantea*—a large species of clam whose soft parts weigh in excess of 300 g in mature adults—showed reduced levels of all pesticides except endrin between 1972 and 1973 (Table 1). Trace amounts of endrin were found in this species in 1973 but not in 1972. The Tombigbee River Basin drains away from the Mississippi River, thus agricultural lands are not as extensive as around the Yazoo and Big Black Basins. As anticipated in this large species from the Tombigbee River Basin, DDT and toxaphene levels were lower in 1972. Also, its residues were proportionately less reduced by 1973 than the residues in other, smaller species from the Delta basins. Since 300 g of tissue was too large an amount to be extracted by this procedure, 20 g of tissue consisting of approximately equal amounts from the gill, mantle, foot, and visceral mass were extracted. Reported levels may have varied slightly because of differences in the amounts of tissues extracted.
In the Pearl River Basin, 1973 residues of DDT and its metabolites in clams and water also decreased in areas where detectable levels had been found the previous year. Toxaphene residues in clams increased slightly (Table 1).

This Pearl River Basin, which is located south of the Delta region and east of the Mississippi River, receives fewer pesticides than the Yazoo, Big Black, or Tombigbee River Basins. The increase in toxaphene residues, although slight, is further evidence of the tendency of clams to reflect local fluctuations since no increases in toxaphene were observed in the flooded areas. Chlordane was found in clams in 1972 at one site on the Pearl River near Jackson, Mississippi (Fig. 1, No. 6), but was not found in 1973. Chlordane residues were very low and no residues of chlordane were detected in any other clam or water samples throughout the state.

Results of analysis of clams from the Pascagoula River Basin were similar to the Pearl River Basin results (Table 1). At Stations 10 and 12 (Fig. 1) slight increases in both toxaphene and DDT and its metabolites were recorded. These increases were between 0.005 µg/g and 0.01 µg/g in all but one sample (Table 1). On the Leaf River, Station 11, a decrease in DDT levels was observed between 1972 and 1973. This basin is the farthest from the Delta region, and residues were so low that no other trends in pesticide changes could be determined between 1972 and 1973.

A comparison of these increases and decreases in DDT residues in clams shows that *C. manilensis* is the best indicator of change. In every instance in 1972, *C. manilensis* contained the highest levels of DDT and toxaphene. In 1973, *C. manilensis* again showed the most substantial reductions (Table 1). This imported species, which is taxonomically, anatomically, and physiologically different from the Unionid species tested, might have a greater tendency to concentrate pesticides and a correspondingly greater ability to eliminate them.

Conclusions

Results indicate that freshwater clams are of value in reflecting changes in pesticide levels in streams. In areas where pesticides have been used for many years, once-a-year sampling programs may be sufficient to reflect changes in extremely residual pesticides, such as DDT. At all sampling sites within the Delta region of Mississippi, large reductions, not elimination, of DDT and its metabolites were recorded in clams between 1972 and 1973. In other areas of the state where pesticides were not expected because of lack of large agricultural operations, none were found; therefore the greatest contributor of pesticides to the streams of Mississippi is agricultural usage.

Although it is an indirect means of assessment, monitoring pesticides in freshwater clams may provide additional information on residue occurrence. Analysis of water is not reflective of biological concentrations.

Different species of clams also seem to have varying tendencies to concentrate and varying abilities to eliminate pesticides. *C. manilensis* is apparently the best indicator of the freshwater species sampled.

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Organochlorine and Heavy Metal Residues in Black Duck Eggs from the Atlantic Flyway, 1978 [1]

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ABSTRACT

Black duck (Anas rubripes) *eggs were collected during 1978 in the Atlantic Flyway. One egg from each of 49 clutches was analyzed for organochlorine compounds and mercury. DDE was detected in 39 eggs, ranging from 0.09 ppm to 3.4 ppm, wet weight. DDE residues were highest in eggs from Delaware, where the mean DDE level was 2.0 ppm. DDT and TDE were present at low levels in only five and four eggs, respectively. PCBs resembling Aroclor 1260 were detected in 24 eggs and ranged from 0.43 ppm to 2.9 ppm. Eggs from Massachusetts and Rhode Island contained an average of >1.0 ppm PCBs, but eggs from Nova Scotia, Pennsylvania, Maryland, and Virginia contained no detectable PCBs. Dieldrin, oxychlordane, and heptachlor epoxide were present in a few samples at low levels. Mercury was detected in 31 eggs, ranging from 0.07 ppm to 0.34 ppm, wet weight. Twenty eggs analyzed for chromium, copper, and arsenic contained averages of 0.64 ppm, 1.7 ppm, and 0.18 ppm, respectively. No geographic pattern was observed in these metal residue levels. Eggshell thickness (0.347 mm) was identical to the pre-1946 norm.*

Introduction

Concern over reports of a declining black duck (*Anas rubripes*) population in the Atlantic Flyway (*7, 11*) led to a survey of organochlorine pesticides in eggs of this species in 1964 (*13*). DDE averaged over 4 ppm in eggs from three states in that survey, and DDT was present in all eggs analyzed. Use of DDT in the United States had declined when another collection of eggs was made in 1971 to see if residues in black duck eggs had also declined (*9*). Although DDT had decreased and there was a general downward trend in DDE residues, individual eggs in 1971 still contained DDE levels comparable to those in 1964.

The egg collection was again repeated in 1978, almost six years after a virtual ban on use of DDT in the flyway. The 1978 survey was undertaken to determine trends in organochlorine pollutant loads, to gain information on heavy metal residues in black duck eggs, and to generally assess the relationship of these residues to population levels of black ducks in the Atlantic Flyway. The results are reported here.

Sample Collection and Preparation

In 1978, biologists in the Atlantic Flyway located and collected two eggs from each of 49 black duck nests. Eggs were individually wrapped in aluminum foil, packed in foam rubber, and shipped via air freight to the Patuxent Wildlife Research Center, Laurel, Maryland.

Egg measurements included weight, maximum length and breadth, and volume. Volume was measured by water displacement if eggs were whole, and by the formula $V = 0.506315LB^2 + 0.924992442$, where V = volume, ml; L = length, cm; B = breadth, cm, if eggs were cracked (*9*). Because dehydration or loss of lipid occurs in embryonated eggs, a specific gravity of 1.0 was assumed for all eggs, and residue values (ppm) were based on egg volume (*16*).

All dirt was cleaned off eggshells before they were opened at the equator and their contents extruded into a glass jar. Stage of embryonic development was noted and the contents were frozen until time for chemical analysis. One egg per clutch was analyzed for organochlorines and mercury (Hg). Twenty of these eggs were also analyzed for copper (Cu), chromium (Cr), and arsenic (As): two from Nova Scotia, three from Maine, three from New Hampshire, two from Massachusetts, four from Rhode Island, four from Delaware, and two from Virginia. Eggshells were washed with membranes intact and air-dried for two weeks. Four shell thickness measurements were taken at the equator with a Starrett 1010M micrometer with 0.01 mm graduations. The mean of these four values was considered the eggshell thickness for that egg.

Statistical comparison of residue levels in 1971 and 1978 were made, where possible, by a Student *t*-test. Since arithmetic and geometric means were nearly equal, arithmetic rather than geometric means were

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compared. Comparison of mean eggshell thickness measurements from pre-1946, 1964, 1971, and 1978 was made with one-way analysis of variance and Bonferroni multiple comparisons.

Analytical Procedures

ORGANOCHLORINES

Eggs were analyzed for *p,p'*-DDE, *p,p'*TDE, *p,p'*-DDT, dieldrin, heptachlor epoxide, oxychlordane, *cis*-chlordane, *trans*-nonachlor, *cis*-nonachlor, endrin, HCB, mirex, toxaphene, and polychlorinated biphenyl (PCB) residues. Egg contents were homogenized, mixed with anhydrous sodium sulfate, and extracted with hexane in a Soxhlet apparatus for 7 hours. Extracts were cleaned on a partially deactivated Florisil column (*1*). Pesticides and PCBs were separated on a SilicAR column into four fractions as described by Kaiser et al. (*8*).

Residues were quantified on a gas-liquid chromatograph equipped with a electron-capture detector and a column packed with a mixture of 1.5 percent OV–17 and 1.95 percent QF–1, under the conditions described by Kaiser et al. (*8*). Residues in three samples were confirmed on a Finnigan 4000 series gas-liquid chromatograph/mass spectrometer (*8*).

Recoveries of pesticides and PCBs from fortified chicken eggs averaged 94 percent. Residues reported here were not corrected for recovery. The lower limit of quantification was 0.1 ppm for all pesticides and 0.5 ppm for PCBs.

MERCURY

Eggs were homogenized in a Virtis blender, and a 5-g portion was taken for Hg analysis. Samples were digested by a method similar to that described by Monk (*12*). Mercury levels were determined by a cold vapor atomic absorption spectrometry method similar to that of Hatch and Ott (*4*), using a Coleman Model MAS 50 mercury analyzer. A portion of sample was added to a BOD bottle and diluted to 50 ml with water. Five milliliters of 10 percent hydroxylamine hydrochloride and 5 ml of 10 percent stannous chloride were added to the mixture to reduce Hg ions to metallic Hg. The sample was aerated and the Hg was measured in the air stream passing through a gas cell in the path of ultraviolet light at 253.7 nm.

Recoveries of Hg from spiked chicken liver samples averaged 93 percent. The lower limit of quantification was 0.02 ppm. Samples were not corrected for percent recovery.

COPPER, CHROMIUM, AND ARSENIC

A 5-g portion of each homogenized egg was dried for 2 hours at 110°C in a drying oven, then charred for 2 hours at 200°C in a muffle furnace. The temperature was raised to 600°C at a rate of 100°C/hour and the sample was left to ash overnight. The ash was cooled, dissolved over a hotplate in approximately 4 ml nitric acid and 1 ml hydrochloric acid, transferred to a 50-ml polypropylene centrifuge tube, and diluted to 10 ml with distilled deionized water. This solution was used for Cu and Cr analyses.

A 5-g aliquot was digested overnight at room temperature in 20 ml concentrated nitric acid. After an addition of 5 ml aqueous 1 percent Ni as $Ni(NO_3)_2$, the acid was slowly boiled away until the volume in the flask was approximately 1–3 ml. The remainder was transferred to a 50-ml polypropylene centrifuge tube and diluted to 25 ml with distilled deionized water. This solution was used for As analysis.

All heavy metal determinations were made by flameless atomic absorption spectrophotometry with a Perkin-Elmer 460 AAS instrument equipped with an EGA 2200 graphite furnace and accessory ramp, a deuterium arc background corrector, an AS 1 autosampler set for 20 μl injections, pyrolytically coated graphite tubes, and a PRS 10 printer. The furnace conditions were as follows:

Dry at 110°C for 40 seconds with 30-second ramp
Char at 900°C for 40 seconds with 30-second ramp
Atomize at 2700°C for 5 seconds with no ramp

The peak height mode was used with an integration time of 7 seconds.

The Cu and Cr determinations were made by comparison with aqueous standards at wavelengths of 324.7 nm and 357.8 nm, respectively, using hollow cathode lamps.

The As determination was made by the method of additions with the matrix modification of adding a 1 percent solution of Ni as $Ni(NO_3)_2$ to the furnace as described by Shum et al. (*14*). An electrodeless discharge lamp and wavelength of 193.7 nm were used.

The lower limits of quantification were 0.05 ppm for Cu and Cr and 0.1 ppm for As. Recoveries from fortified chicken livers ranged from 92 percent to 97 percent; residues were not corrected on the basis of these data.

Results and Discussion

ORGANOCHLORINES

DDE and PCBs were the only organochlorines detected in the majority of the 49 eggs analyzed (Table 1). DDE was present in 39 eggs; the average concentra-

TABLE 1. *DDE, PCB, and mercury residues in black duck eggs from the Atlantic Flyway, 1978*

Location Collection	No. of Clutches	*p,p'*-DDE	Aroclor 1260	Hg
CANADA				
Nova Scotia	3	0.32 ± 0.15[1] ND–0.58 (2)[3]	ND[2]	0.13 ± 0.03 0.07–0.16 (3)
New Brunswick	1	ND	0.60 (1)	0.14 (1)
Quebec	4	0.64 ± 0.45 0.19–2.0 (4)	0.25 ± 0.20 ND–0.84 (1)	0.12 ± 0.04 ND–0.21 (3)
UNITED STATES				
Maine	6	0.26 ± 0.19 ND–1.2 (4)	0.34 ± 0.29 ND–1.8 (1)	0.18 ± 0.05 ND–0.34 (5)
New Hampshire	5	0.32 ± 0.15 0.12–0.96 (5)	0.61 ± 0.31 ND–1.7 (3)	0.11 ± 0.03 ND–0.20 (4)
Vermont	4	0.44 ± 0.25 0.14–1.2 (4)	0.44 ± 0.23 ND–0.95 (2)	0.10 ± 0.01 0.08–0.14 (4)
Massachusetts	2	0.63 ± 0.37 0.26–1.0 (2)	1.1 ± 0.10 1.0–1.2 (2)	0.08 ± 0.01 0.07–0.09 (2)
Rhode Island	5	0.36 ± 0.05 0.25–0.50 (5)	1.3 ± 0.21 0.62–1.8 (5)	0.13 ± 0.04 ND–0.25 (4)
Pennsylvania	1	0.11 (1)	ND	0.08 (1)
Delaware	10	2.0 ± 0.25 0.84–3.4 (10)	0.98 ± 0.23 ND–2.9 (9)	0.09 ± 0.02 ND–0.30 (4)
Maryland	2	0.10 ± 0.02 0.09–0.12 (2)	ND	ND
Virginia	6	ND	ND	ND
Atlantic Flyway	49	0.65 ± 0.12 ND–3.4 (39)	0.56 ± 0.09 ND–2.9 (24)	0.11 ± 0.01 ND–0.34 (31)

[1] Arithmetic mean ± SE. Range of values appears under the mean.

[2] ND—Sample or samples analyzed were below the quantification limit of 0.1 ppm before correction for moisture loss. If no eggs from a sampling unit contained quantifiable residues, ND appears in table. If one or more eggs from the sampling unit contained quantifiable residues, ND samples were used in calculating means as 0.05 ppm. ND samples in flyway means also equaled 0.05 ppm.

[3] Numbers in parentheses are the number of samples containing quantifiable residue levels.

tion was 0.65 ppm DDE and the highest concentration was 3.4 ppm. Eggs from New Brunswick and Virginia contained no DDE. In the nine states and provinces where eggs were collected in both 1971 and 1978, DDE in eggs averaged 1.2 ppm and 0.80 ppm, respectively ($P > 0.05$). Eggs collected in Delaware in the 1971 survey averaged 5.9 ppm DDE and ranged up to 14 ppm. Eggs in the present survey averaged only 2.0 ppm DDE, a significant decrease ($P < 0.05$), and the highest residue level was 3.4 ppm. In contrast, eggs from Vermont contained comparable levels of DDE in 1971 and 1978. Mean DDE levels were 0.51 ppm in 1971 and 0.44 ppm in 1978; the highest DDE level detected was 1.0 ppm in 1971 and 1.2 ppm in 1978. It would appear that although states with higher DDE levels in black duck eggs have shown decreases over the last seven years, those states with lower levels are still showing little decline.

The incidence of DDT residues in 1971 and 1978 samples varies significantly. DDT occurred at > 0.1 ppm in 31 of 61 eggs analyzed in 1971, but in only 5 of 49 eggs analyzed in 1978. Three of these eggs were collected in Delaware (0.09–1.3 ppm DDT) and two in Rhode Island (0.14 ppm and 0.17 ppm).

Low levels (0.10–0.15 ppm) of TDE were found in only four of the 49 eggs analyzed in 1978 (two from Delaware and one each from Maine and Quebec). In 1971, TDE was detected at levels > 0.1 ppm in 11 of the 61 eggs.

PCBs resembling Aroclor 1260 were detected in 24 of the 49 black duck eggs. They were not detected in eggs from Nova Scotia, Pennsylvania, Maryland, and Virginia. Average PCB values ranged from 0.25 ppm in Quebec to 1.3 ppm in Rhode Island. The highest PCB residue of 2.9 ppm was detected in an egg from Delaware. These residues cannot be quantitatively compared with 1971 residues because a different analytical technique was used; however, residues were generally lower and occurred in a smaller percentage of samples.

Dieldrin was found at low levels in ony two samples analyzed, one from Delaware (0.09 ppm) and one from Massachusetts (0.17 ppm). Heptachlor epoxide was detected in only one sample, collected in Rhode Island (0.14 ppm). Again these are not comparable to 1971 residue levels. The decreased incidence of residues is related to the increase of detection limit from 0.05 ppm to 0.1 ppm for dieldrin and from 0.01 ppm to 0.1 ppm for heptachlor epoxide. Dieldrin was detected at levels > 0.1 ppm in 13 of the 61 eggs analyzed in 1971 and heptachlor epoxide was present in only two of the 1971 eggs at this level. These residue levels cannot be quantitatively compared because of differences in analytical techniques, but low concentrations were observed in eggs from both surveys. Oxychlordane was present in one black duck egg from Maine (0.12 ppm), two eggs from New Hampshire (0.13 and 0.16 ppm), one egg from Massachusetts (0.08 ppm), and one egg from Maryland (0.05 ppm). No other organochlorine residues were detected.

HEAVY METALS

Mercury was detected in 31 of the 49 eggs analyzed (Table 1). The overall mean was 0.11 ppm Hg, and residues ranged up to 0.34 ppm. No Hg was detected in eggs from the two states, Maryland, and Virginia. Other state and province means ranged from 0.08 ppm in Massachusetts and Pennsylvania to 0.18 ppm in

Maine. These levels are below those usually associated with decreased hatchability or survival of young pheasants (*2, 15*) or mallards (*5, 6*). Captive black ducks on a diet of 3 ppm methylmercury produced eggs containing 4–6 ppm Hg, wet weight, and both hatching and survival of young were depressed (*3*). The residues observed in eggs from the 1978 field collection are below this effect level.

Chromium and copper were detected in all 20 eggs analyzed for heavy metals. Average residue levels (mean ± SE) were 0.64 ± 0.07 ppm Cr and 1.68 ± 0.10 ppm Cu. Arsenic was present in 18 of the 20 eggs analyzed; the average level was 0.18 ± 0.02. Eggs from different sampling sites contained comparable Cu, Cr, and As residues.

EGGSHELL THICKNESS

Eggshell thicknesses of black duck eggs collected before 1946, when DDT use became widespread, and in 1964, 1971, and 1978 are presented in Table 2. Eggshell thickness in 1978 eggs averaged 0.345 mm (N = 49 clutches). Eggs more than 1-week developed were eliminated from the analyses, since embryo development can affect eggshell thickness. The 36 clutches remaining showed an average thickness (mean ± SE) of 0.347 ± 0.003 mm. These eggs were used in comparing 1978 eggshell thickness to thickness during other years. The range of eggshell thickness in 1978 was 0.279–0.389 mm.

There was no difference in eggshell thickness between eggs collected before 1946 (0.347 mm) and those collected in 1978 (0.347 mm). Eggshells collected in 1971 were also of a thickness comparable to those collected before 1946 and during 1978. Eggshells collected in 1964, however, were 7.5 percent thinner than the pre-1946 norm, and significantly thinner ($P <$ 0.01) than eggshells collected in 1971 and 1978. This decrease in 1964 eggshell thickness was perhaps associated with the higher DDE residues found in the black duck eggs during that 1964 survey. DDE has been shown to cause shell thinning and lowered reproductive success in captive black ducks (*10*).

TABLE 2. *Shell thickness of black duck eggs from the Atlantic Flyway*

Parameter	Date of Collection			
	Pre-1946	1964	1971	1978
Eggshell thickness[1] (mm ± SE)	0.347 ± 0.003[2]	0.321 ± 0.004[3]	0.343 ± 0.003[3]	0.347 ± 0.003
N	38[4]	37[5]	52[5]	36[5]
% decrease from pre-1946	—	7.5[6]	1.2	—

[1] Mean of four measurements at the equator, membranes intact, all eggs developed < 7 days.
[2] Data are from H. M. Ohlendorf and E. E. Klaas, U.S.F.W.S., personal communication, July 1979.
[3] Data are from Longcore and Mulhern (*9*) and J. R. Longcore, U.S.F.W.S., personal communication, July 1979.
[4] N = number of clutches from which all eggs were measured and a clutch mean determined.
[5] N = number of clutches, from which one or two eggshells were measured.
[6] Eggshell thickness is lower than in all other years, one-way analysis of variance, Bonferroni pair-wise comparisons, $P < 0.01$.

Summary

DDE and PCBs were the only organochlorines found routinely in black duck eggs collected in 1978. DDE had not decreased significantly from 1971 levels on a flyway basis, but residues in more heavily contaminated areas had decreased. Mercury was found at low (0.1–0.3 ppm) levels in the eggs and chromium, copper, and arsenic were detected at levels below 2.5 ppm. Eggshell thickness was comparable to pre-1946 measurements; organochlorine and mercury residues were below those associated with reproductive problems in captive waterfowl.

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Significance of Organochlorine and Heavy Metal Residues in Wintering Shorebirds at Corpus Christi, Texas, 1976–77

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ABSTRACT

Organochlorine and heavy metal residues were determined in 103 shorebirds of seven species collected at Corpus Christi, Texas, during the winter of 1976–77 to evaluate their potential effects on population survival. DDE and polychlorinated biphenyls (PCBs) were detected in most samples. Chlordane isomers, dieldrin, toxaphene, and heptachlor epoxide also occurred, but less frequently. In general, organochlorine residues were low in skinned carcasses. Geometric means on a wet weight basis ranged from 0.25 ppm to 4.76 ppm for DDE and from 0.67 ppm to 6.64 ppm for PCBs; residues of the other compounds averaged less than 1 ppm in all instances. Mercury, lead, arsenic and vanadium occurred in all shorebird livers, and selenium and cadmium were detected in all kidneys. Residues of these metals, except selenium, were low in most tissue samples. Selenium averages varied from 1.77 ppm to 5.62 ppm (wet weight) in kidneys; residues in this range may be sufficient to inhibit reproduction or to induce other forms of toxicity, especially at the higher levels.

Introduction

During the winter, large numbers of shorebirds of many species use the mudflats and shallow inlets of the bays at Corpus Christi, Texas. The south shore of Nueces Bay is crowded with an industrial complex including oil refineries, a zinc-smelting plant, chemical plants, and other industries; agricultural lands encompass much of the northern shores of Nueces and Corpus Christi Bays (Fig. 1). Sediments from parts of the Bay system are heavily contaminated with zinc and cadmium (*9*), and eggs of some aquatic birds have high levels of DDE (D. H. White, unpublished data). Thus, the potential for pollution problems is high in this area because of industrial effluents and pesticide runoff from surrounding croplands. Conservationists have expressed concern that wintering birds in this area may be accumulating harmful levels of environmental contaminants. This study was conducted to determine the levels of organochlorine and heavy metal residues in tissues of wintering shorebirds and to evaluate the significance of contamination on population survival.

Collection Methods

Shorebirds were collected at opportune times from December 1976 through March 1977 at the locations listed by species on Figure 1. Birds were killed using 12-gauge #4 steel shot so that tissue analyses for lead would not be biased. Soon after collection, birds were tagged, placed individually in polyethylene bags, and stored frozen until they were prepared for analysis. A total of 103 shorebirds of seven species was collected for organochlorine and heavy metal analysis.

Analytical Procedure

Organchlorine analyses were conducted at the Patuxent Wildlife Research Center, Laurel, Maryland. Before analysis, the birds were skinned, and the feet, beaks, wingtips, and gastrointestinal tracts were removed. The kidneys and livers were saved for heavy metal analyses. The carcasses were homogenized in a food grinder and a 10-g portion was mixed with anhydrous sodium sulfate and extracted 7 hours with hexane in a Soxhlet apparatus. Lipids were removed by Florisil column chromatography. The procedures used were those described by Cromartie et al. (*3*), except that the organochlorines were separated into four fractions on the SilicAR column rather than three fractions to ensure the separation of dieldrin or endrin into an individual fraction (*10*).

The pesticides in each fraction were identified and quantified with a gas-liquid chromatograph equipped with an electron-capture detector and a 1.5 percent OV–17/1.95 percent QF–1 column. Recoveries of pesticides and PCBs from spiked samples averaged 94 percent; residues were not corrected for recovery. The lower limit of quantification was 0.1 ppm for all pesticides and 0.5 ppm for PCBs. All residues are expressed as ppm wet weight. Residues in 13 percent of

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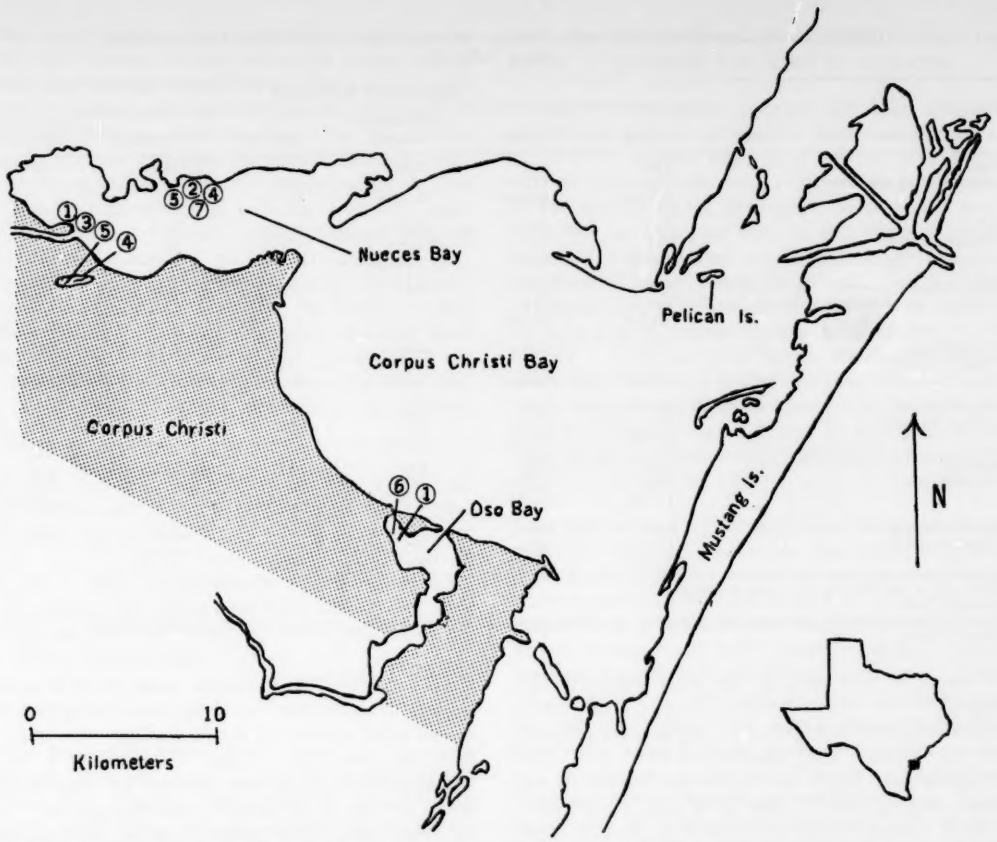

FIGURE 1. *Shore bird collection sites at Corpus Christi, Texas, 1976–77. Circled numbers represent sites for separate species; 1 = avocet, 2 = dunlin, 3 = greater yellowlegs, 4 = least sandpiper, 5 = lesser yellowlegs, 6 = sanderling, and 7 = western sandpiper.*

the samples were confirmed with a Finnigan 4000 series gas-liquid chromatograph/mass spectrometer.

Shorebird livers and kidneys were analyzed for certain heavy metals at Analytical Bio Chemistry Laboratories, Inc., Columbia, Missouri. Samples were shipped frozen in dry ice in chemically cleaned jars.

Samples were digested with 8 ml concentrated, double-distilled nitric acid for 4 hours on a reflux apparatus. Water condensers were used to condense the nitric acid during the digestion. Following digestion, the samples were diluted to 50 ml with 1 percent hydrochloric acid. A subsample of this digestate was further digested with nitric and nitric:perchloric acid to white fumes of perchloric acid. The sample digestate was then diluted to 25 ml with deionized water. The sample was then analyzed by atomic absorption or flame emission spectrophotometry. When graphite furnace techniques were used, solution concentrations were determined by the method of additions. Solution concentrations of those elements where flame techniques were used were determined by comparison of sample and standard peak heights. The standards contained approximately the same acid concentrations as the samples.

Analyses were conducted on a PE 305B atomic absorption unit with background correction. Graphite furnace analysis was performed on a PE MHS–10 hydride generation system. Recoveries of the various metals from spiked samples ranged from 74 percent to 106 percent; residues were not corrected for recovery. The lower limit of quantification was 0.1 ppm for mercury, lead, arsenic, selenium, and cadmium, and 0.05 ppm for vanadium. All residues are expressed as ppm wet weight.

Results and Discussion

ORGANOCHLORINE RESIDUES

Residues of DDE, PCBs, chlordane isomers, dieldrin, toxaphene, and heptachlor epoxide in shorebird carcasses are shown in Table 1. Mirex was present in only

TABLE 1. *Organochlorine residues in carcasses of wintering shorebirds from Corpus Christi, Texas, 1976–77*

Species	No.	Residues, ppm Wet Weight					
		DDE	PCBs	Chlordane Isomers	Dieldrin	Toxaphene	Heptachlor Epoxide
Avocet (*Recurvirostra americana*)	4	1.49[1] (4)[2] 0.5–2.6[3]	0.79 (4) 0.1–3.0	0.96 (2) 0.7–1.4	0.47 (3) 0.2–1.0	ND	ND
Dunlin (*Calidris alpina*)	13	3.15 (13) 1.0–9.0	1.19 (13) 0.5–8.0	0.12 (2) —	0.19 (7) 0.1–0.4	0.21 (5) 0.1–0.4	ND
Greater Yellowlegs (*Tringa melanoleucus*)	2	1.90 (2) 1.5–2.4	0.97 (2) 0.8–1.2	ND	ND	ND	ND
Least Sandpiper (*Calidris pusillus*)	30	1.07 (30) 0.2–4.6	0.67 (16) 0.8–3.6	0.16 (3) 0.1–0.7	0.16 (14) 0.1–0.6	0.19 (11) 0.1–0.5	0.32 (2) 0.2–0.5
Lesser Yellowlegs (*Tringa flavipes*)	17	4.55 (17) 1.7–9.0	0.79 (14) 0.5–1.8	0.13 (3) 0.1–0.2	0.16 (9) 0.1–0.3	0.29 (9) 0.2–0.7	0.14 (2) 0.1–0.2
Sanderling (*Calidris alba*)	15	4.76 (15) 1.6–10	6.64 (15) 2.4–18	0.66 (15) 0.2–1.7	0.28 (13) 0.1–0.6	0.14 (1) —	0.19 (14) 0.1–0.3
Western Sandpiper (*Calidris mauri*)	22	0.25 (17) 0.1–0.7	0.95 (22) 0.2–4.3	0.20 (7) 0.1–0.4	0.13 (8) 0.1–0.2	0.24 (9) 0.1–1.9	ND

NOTE: ND = not detected; — = not applicable.
[1] Geometric mean.
[2] Values in parentheses are actual number of samples containing detectable residues. These values were used in calculating means.
[3] Extreme values.

one sample at 0.53 ppm, but no other organochlorine compounds were detected in these samples. DDT metabolites were present in 95 percent of the samples analyzed, with most residues being identified as DDE. DDE concentrations were highly variable among individuals and species, ranging from 0.1 ppm to 10 ppm. Lowest overall DDE concentrations were found in western sandpipers (*Calidris mauri*) and the highest occurred in sanderlings (*Calidris alba*) (Table 1).

In general, DDE residue concentrations in shorebird carcasses were relatively low, and only moderately higher than levels measured in control birds in dietary experiments. Mallards (*Anas platyrhynchos*) fed diets containing 40 ppm DDE for 96 days averaged 33 ppm in carcasses 42 days after cessation of treated food; 11 months after DDE exposure, carcasses averaged 9.6 ppm in treated birds and only 0.5 ppm in controls (*8*). Black ducks (*Anas rubripes*) fed 10 ppm DDE for 7 months averaged 155 ppm in carcasses whereas controls averaged about 0.3 ppm (*12*); 2 years after exposure ceased, DDE levels in black ducks averaged 12 ppm in males and 3.4 ppm in females. Thus, it appears that shorebirds in the area sampled are being exposed to low environmental levels of DDE, if we assume that residue accumulation and loss is directly correlated with exposure (*20*).

PCBs occurred in 83 percent of the samples, ranging from 0.1 ppm to 18 ppm. As with DDE, PCB residue levels were highly variable among individuals and species (Table 1). The geometric mean of PCB concentrations in sanderlings was about seven-fold higher than in any other species; all the sanderlings were collected from Oso Bay (Fig. 1) near the outlet of a sewage treatment plant which may account for their elevated levels. Overall, PCB levels in shorebird carcasses were far below those levels known to cause death or reproductive problems in other avian species (*19*). Carcasses of great cormorants (*Phalacrocorax carbo*) suspected of dying from PCB poisoning contained residue concentrations ranging from 29 ppm to 460 ppm (*11*). Because PCBs are highly cumulative in avian tissues, as is DDE, we believe that shorebirds in the Corpus Christi area probably are being exposed to low environmental levels of these compounds.

Dieldrin occurred in only 52 percent of the samples and levels averaged less than 0.5 ppm in the 6 species where it was found (Table 1). Although dieldrin is much more toxic to birds than is DDE or PCBs, the present levels in shorebirds are not considered to be in the danger zone. Residue levels in carcasses of birds known to have died from dieldrin poisoning in experimental studies ranged from about 10 ppm to 36 ppm (*23*), much higher than the carcass levels reported in our study.

Chlordane metabolites including oxychlordane, *cis*-chlordane, *trans*-nonachlor, and *cis*-nonachlor were found in 32 percent of the samples, mostly in sanderlings (Table 1). Residues of all metabolites were low,

so they were combined for convenience and reported as *chlordane isomers*. Combined residue concentrations in individual samples ranged from 0.1 ppm to 1.7 ppm. All the sanderlings and 2 avocets (*Recurvirostra americana*) were collected from Oso Bay (Fig. 1) and this group of samples contained the highest chlordane residue levels; as stated earlier, the collection site in Oso Bay was near the outlet of a sewage treatment plant and the residues probably reflect domestic use of chlordane on lawns and gardens. Oxychlordane was the most persistent and the most toxic chlordane metabolite in bird-feeding studies (*21*). The levels in bodies of birds that died and in survivors were far above those reported in this study; therefore, the present levels are not suspected of contributing to shorebird mortality. The sublethal effects of chlordane to birds are unknown (*21*).

Heptachlor expoxide was present in only 17 percent of the samples and, as with other chlordane metabolites, occurred mostly in sanderlings (Table 1). Heptachlor epoxide found in bird tissues may be the result of exposure to either heptachlor or chlordane (*21*). Its adverse effects on wildlife have been documented (*20, 1*); however, the low levels we report for shorebirds are probably insignificant.

HEAVY METALS

Rsidues of certain heavy metals found in shorebird tissues are shown in Table 2. Livers were analyzed for mercury, lead, arsenic, and vanadium and kidneys were analyzed for selenium and cadmium. All the metals determined are known to be toxic to birds in sufficient quantities. All occur naturally in the environment; however, the burning of fossile fuels and many industrial, domestic, and agricultural uses of these elements contribute to abnormally high levels in some areas.

In dietary experiments, mercury has been shown to accumulate greatest in feathers, liver, and kidney (*7, 22*); uptake is rapid and loss is slow. A large proportion of the total body burden of mercury is eliminated in the feathers during molt (*22*). Most of the shorebirds that we analyzed were in full winter plumage; therefore, liver residues should give a fair indication of recent exposure levels. In general, mercury levels in our samples were considerably lower than those reported for some aquatic birds in other studies (*5, 6*), but were similar to those in shorebirds from a relatively unpolluted area in England (*16*). Mercury levels in our samples ranged from 0.05 ppm to 5.45 ppm, but most were below 0.5 ppm (Table 2). It is believed that these levels are below those suspected of causing toxic effects in other birds (*6*).

Lead residues in liver are considered indicative of current lead exposure, and levels that range between 6 ppm and 20 ppm (wet weight) or greater may be diagnostic of acute intoxication, at least in waterfowl (*13*). Only 3 birds in our study, all western sandpipers, were within this range and residue levels in all but 2 species averaged less than 0.2 ppm (Table 2). However, Dieter (*4*) suggests that birds with liver lead levels between 0.5 ppm and 1 ppm (0.86 ppm average) may exhibit abnormally low activity of plasma delta-aminolevulinic acid dehydratase, an enzyme important in the synthesis of hemoglobin and specifically inhibited by lead. Thirty-six percent of our samples contained lead residues within that range; sanderlings and western sandpipers had the highest residues. It is unknown whether these levels adversely affect shorebirds.

TABLE 2. *Heavy metal residues in livers and kidneys of wintering shorebirds from Corpus Christi, Texas, 1976–77*

Species	No.[1]	Residues, ppm Wet Weight					
		Mercury	Lead	Arsenic	Vanadium	Selenium	Cadmium
Avocet	4	0.22[2] 0.05–0.53[3]	0.11 0.05–0.19	0.10 0.05–0.26	0.02 0.02–0.05	2.60 2.20–3.90	1.82 0.79–8.20
Dunlin	10	0.47 0.28–0.78	0.19 0.05–0.58	0.07 0.05–0.35	0.06 0.03–0.16	3.55 2.40–5.00	1.94 0.52–7.30
Greater Yellowlegs	2	0.83 0.67–1.04	0.11 0.05–0.25	0.05 —	0.03 0.02–0.04	1.77 1.30–2.40	1.47 1.20–1.80
Least Sandpiper	16	0.50 0.11–1.69	0.17 0.05–1.28	0.06 0.05–1.50	0.07 0.03–0.23	4.23 1.60–6.90	4.31 0.83–22.7
Lesser Yellowlegs	13	0.21 0.11–0.73	0.18 0.05–1.17	0.10 0.05–0.42	0.04 0.02–0.16	2.43 1.50–4.20	1.33 0.44–2.60
Sanderling	15	0.67 0.12–5.45	0.93 0.24–2.01	0.14 0.05–1.23	0.07 0.02–1.20	3.96 2.10–6.40	1.38 0.33–7.90
Western Sandpiper	15	0.43 0.20–0.86	0.74 0.05–28.5	0.23 0.05–1.10	0.10 0.03–0.58	5.62 2.80–10.2	2.74 0.66–12.0

[1] All samples analyzed contained detectable levels of each metal. Livers were analyzed for mercury, lead, arsenic, and vanadium; kidneys were analyzed for selenium and cadmium.
[2] Geometric mean.
[3] Extreme values.

Arsenic is a major by-product of the smelting industry and a component of herbicides and defoliants used in agriculture (*14, 17*), thus the potential for abnormal arsenic accumulation in the environment appeared to be present in the Corpus Christi area. However, residue levels in our samples were quite low overall, averaging less than 0.3 ppm in all instances and ranging up to 1.5 ppm (Table 2). The significance of these low levels in shorebirds is unknown, but the levels probably reflect background contamination and are not suspected of being extremely harmful. Blus et al. (*2*) reported similar arsenic levels in livers of South Carolina brown pelicans (*Pelecanus occidentalis*).

Vanadium concentrations in livers were lowest of all the metals; residues averaged less than 0.1 ppm in most species (Table 2). White and Dieter (*24*) found that very little vanadium accumulated in tissues of mallard ducks fed up to 100 ppm vanadium in the diet; although vanadium accumulated in small amounts, tissue levels increased with treatment level. The liver levels we report here are similar to those reported for control birds in that study and are not suspected of being particularly harmful, overall. However, lipid metabolism may be altered in laying females when vanadium in the liver approaches 0.5 ppm (*24*). Several of our birds had levels of this magnitude.

Selenium levels in shorebird kidneys (Table 2) appeared to be abnormally high when compared with the results of a study involving chickens. Laying hens fed 5, 7, and 9 ppm selenium in the diet accumulated on the average about 2.5 ppm in the kidney while controls averaged less than 1 ppm (*15*). Reproduction was inhibited, especially in those hens fed 7 ppm and 9 ppm; significant decreases occurred in egg weight, egg production, and hatchability. Mean kidney levels in our study varied from 1.77 ppm to 5.62 ppm and ranged up to 10.2 ppm in individual samples (Table 2). Body burdens of this magnitude may be sufficient to impair reproduction in shorebirds as well, or cause other forms of toxicity. Since selenium is a by-product of the smelting industry, it is not surprising that levels in shorebirds from the Corpus Christi area appear abnormally high.

Cadmium residue levels in kidneys were highly variable in individual samples, ranging from 0.33 ppm to 22.7 ppm (Table 2). Mean levels were similar to those reported for ruddy ducks (*Oxyura jamaicensis*) and canvasbacks (*Aythya valisineria*) from the Chesapeake Bay area (*26, 28*) and are considered to be fairly low. Kidneys of mallards fed 2 ppm dietary cadmium contained an average of 1.5 ppm and 2.9 ppm after 30 days and 60 days, respectively (*25*); these levels also are similar to what we report here for shorebirds. Two hundred ppm dietary cadmium produced kidney lesions and inhibited spermatogenesis in adult mallards (*27*), but kidney cadmium levels averaged almost 100 ppm in these birds.

Conclusions

Shorebirds from the Corpus Christi area contained relatively low levels of each of the organochlorine compounds detected; experimental studies with other avian species suggest that these residues, in most instances, probably are below levels known to have an adverse effect on avian reproduction and survival. Some samples, however, did contain amounts of DDE or PCBs that approximate the range in which adverse effects may be expected. More important, shorebirds with fairly high residue levels may represent a potential hazard to peregrine falcons (*Falco peregrinus*) and other raptors that occasionally prey on them (*18*). Most residues of mercury, lead, arsenic, vanadium, and cadmium also were below known-effect levels. In contrast, selenium residues in shorebird tissues are cause for concern, because residues in most birds were within or above the range known to inhibit reproduction in chickens. In addition, the interactions of organochlorine and heavy metal residues in wild birds are poorly understood; birds with elevated total body burdens of a mixture of pollutants may be more susceptible to disease, stress, and predation than birds with low levels.

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HUMANS

Overview of Human Exposure to Dieldrin Residues in the Environment and Current Trends of Residue Levels in Tissue

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ABSTRACT

An overview of available literature indicates that dieldrin residues are still found routinely in soil, air, water, and food, despite the 1974 U.S. Environmental Protection Agency ban on the use of aldrin and dieldrin. Dieldrin residue levels in environmental substrates, which are indicative of aldrin or dieldrin use, have decreased significantly since the mid-1960s, the peak usage years. However, human tissue studies do not show a corresponding decline in dieldrin residue levels. Thirteen studies, conducted between 1963 and 1976, show that average dieldrin levels in human adipose tissue and human milk fat remain between 0.160 ppm and 0.220 ppm. Other studies suggest that an equilibrium exists in the distribution of dieldrin among various tissues in humans, including blood, fat, brain, and liver. This relationship indicates that the concentration of dieldrin in any tissue may be used as an index of total body burden. Thus it appears that the concentration of dieldrin in the human body has reached a constant level at which the amount ingested and absorbed equals the amount metabolized and excreted. The mechanism of the stable concentrations is unknown, as are the possible health effects of chronic, low-level exposure to dieldrin.

Since the original inquiries into organochlorine toxicity and persistence in the 1960s, hundreds of studies have been conducted to determine the presence and effects of these pesticides in the environment and in humans. Organochlorines are unusually persistent due to the stability of their basic structural feature, the chlorinated ring. In virtually every study of residues in soil, air, water, and human tissue, two chemicals have stood out as particularly widespread: DDT and dieldrin. The U.S. Environmental Protection Agency (EPA) banned DDT for commercial use in the United States in 1972, and residue levels have since declined significantly (*26*). Dieldrin was banned in 1974. Despite this action, human exposure to dieldrin has not ended. Dieldrin residues are still found routinely in soil, air, water, and food.

Dieldrin reaches soil either directly, through intentional application, or indirectly, through rain or fallout from the air. Once incorporated with soil particles, dieldrin becomes so tightly bound that penetration through soil is negligible (*16*). Adsorption is determined primarily by soil type, although pH, temperature, and moisture content of the soil are also involved. Adsorption is strongest in soils with high clay or organic matter content. The high cation exchange capacity causes dieldrin to adsorb to negatively charged sites on the minerals (*21*). Lateral movement is insignificant, except under conditions of heavy erosion. Residues are stabilized, but they are not readily degraded. The half-life of dieldrin residues in soil is estimated to be 2.8 years, or eight years for 95 percent disappearance (*3*). Measurable residues are frequently apparent 11 years after original use. One study showed 10 percent of a subsoil application remaining 21 years after treatment (*2*). This tenacity is due in part to the paucity of soil organisms capable of decomposing dieldrin. To date, only 10 have been identified.

Calculations using the half-life given above and the annual sales data given in Table 1 indicate that if all dieldrin sold each year from 1960 to 1974 had been distributed evenly over all agricultural acreage in the United States, 0.074 pounds per acre would have accumulated in the soil by 1974—more than five times the amount actually applied that year.

Dieldrin residues circulate constantly from one storage medium to another. Although dieldrin adsorbs tightly to soil particles, small amounts may volatilize immediately after application, thus entering the atmosphere. Even slow volatilization from the tremendous surface area of treated soil and plants may contribute signifi-

[1]U.S. Environmental Protection Agency, Office of Pesticide Programs, Benefits and Field Studies Division (TS/768-M), Washington, D.C. 20460.

TABLE 1. *Domestic sales of aldrin and dieldrin (1,000 pounds), 1960–76*

Year	Aldrin	Dieldrin	Total	Literature Cited
1960	8,109	2,650	10,759	(46)
1961	9,926	2,764	12,690	(46)
1962	10,886	2,990	13,876	(46)
1963	12,152	2,685	14,837	(46)
1964	12,693	2,052	14,735	(46)
1965	14,278	1,814	16,092	(46)
1966	19,327	1,908	21,235	(46)
1967	18,092	1,473	19,565	(46)
1968	13,690	1,332	14,022	(46)
1969	9,902	1,206	11,108	(46)
1970	8,909	749	9,658	(46)
1971	11,615	705	12,320	(46)
1972	11,868	740	12,608	(46)
1973 (est.)	10,000	576	10,576	(46)
1974 (usage)	12,398	283	12,681	(45)
1976 (agr. usage only)	900		900	(15)

cantly to ambient air concentrations. After adsorption, the soil/dieldrin complex can be lifted and transported by wind erosion. These losses, however, are probably small compared to losses during application. Pesticide drift during treatment seems to be the primary factor influencing the transfer of dieldrin from soil to air (*14*).

Airborne pesticides have been detected near agricultural operations, in rural areas, at urban locations, and in wilderness regions many thousands of kilometers from known sources of contamination (*28*). The earliest indication that pesticide residues existed in the ambient air came from the analysis of air samples collected from four California sites during 1963 (*47*). Air monitoring conducted from 1970 to 1972 revealed that dieldrin was present in 94 percent of 2,479 samples taken nationwide, with a mean concentration of 1.6 ng/cu.m (*27*). Dieldrin levels as high as 29.7 ng/cu.m have been recorded at rural locations (*41*). The estimated residence time, i.e., rate of removal by dry deposition or rainfall, is 28 weeks (*1*).

Transport through air is probably the most important factor in the appearance of pesticide residues throughout the world. The detection of dieldrin residues at such remote sites as Barbados (*38*), the island of Bimini (*7*), and Antarctica (*43*) suggests that there may be no part of the troposphere that remains free of residues transported from distant sources.

Once in the air, dieldrin will eventually be deposited onto soil and surface water by rainfall or dust fallout. Through this process of deposition, erosion, and runoff, residues move through the hydrologic cycle. Rivers, lakes, and oceans act as sinks for pesticide residues collected through surface and underground runoff. The half-life of dieldrin in one cubic meter of surface water, that is, the time required for half the original deposit to be lost to the atmosphere, was found to be 1.5 years (*31*), or six years for 95 percent disappearance. In a 1964–68 survey of pesticides in water, dieldrin dominated pesticide occurrences in all regions. It appeared in 39 percent of the samples (*29*). Several samples actually exceeded the environmental limit recommended by the Committee on Water Quality Criteria (*36*). In a second study, dieldrin was present in 40 percent of more than 500 drinking water samples taken from the Mississippi and Missouri Rivers (*40*). The extensive distribution of dieldrin in aquatic systems is indicative of environmental loading from nonpoint sources such as rainfall, dust fallout, and erosion, as well as from agricultural point sources (*36*).

Eventually, the small amounts of dieldrin in soil, air, and water collect in the plant and animal tissues that are food for humans. Dieldrin can be removed from the soil by various root crops, including sugar beets, carrots, radishes, and potatoes (*30*). Soybeans and peanuts, high in oil, may also contain residues (*4*). Lower concentrations are found in some cereal crops, corn, oats, barley, and alfalfa (*19*).

Food is probably the primary source of dieldrin residues in human adipose tissue. The Total Diet Studies of the Food and Drug Administration (FDA), U.S. Department of Health and Human Services provide exhaustive information on levels and exact dietary sources of many persistent pesticides. Dietary intake in the Total Diet Studies is based on the total food consumpton of a 15- to 20-year-old male. Unlike DDT, dieldrin concentrations do not differ with age, sex, or race (*13*), so this information applies to the general population.

According to the 1974 diet study, measurable dieldrin residues (greater than 1 ppb) occurred predominantly in meat, fish, and poultry (100 percent positive) and dairy products (97 percent positive) (*18*). This is due to the lipophilic properties of dieldrin. To avoid bioaccumulation of dieldrin residues, animal feed must be carefully monitored. Residues of 1 ppm in soil may be too high for crops destined for animal consumption (*19*). Garden fruits (60 percent positive), potatoes (30 percent positive), leafy vegetables (10 percent positive), oils and fats (10 percent positive), and fruits (7 percent positive) also contribute to man's daily intake.

The health effects of chronic low-level exposure to dieldrin are unknown. It is tremendously difficult to predict future effects on public health. Dieldrin is known to have carcinogenic, teratogenic, and reproductive effects on test animals (46). In one study, autopsies showed significantly elevated concentrations of dieldrin in patients who had suffered from portal cirrhosis, carcinoma of various tissues, and hypertension (*37*). Dieldrin is transferred to the fetus during pregnancy,

and the child receives additional doses after birth either from its mother's milk or from dairy products (*5*). Thus babies born after organochlorines came into common use may have stored substantial amounts of dieldrin within the first few months of life. Children and young animals are often more susceptible than adults to poisons, drugs, and carcinogens (*20*). The extreme persistence of dieldrin residues in the environment and in mammalian tissue may also cause health problems indirectly. In one case, dairy cattle, fed on hay from a field previously used as a cherry orchard to which dieldrin had been applied, produced milk exceeding acceptable dieldrin levels. The milk was withheld from market for three months until the residues dropped to acceptable levels (*34*).

The purpose of this study was to consolidate information from relevant reports on dieldrin residues in humans regarding the quantitative history of residue sources and concentrations, and to compare this information to the findings of EPA's *National Study to Determine Levels of Chlorinated Hydrocarbon Insecticides in Human Milk, 1975–76*. Data describing human exposure to dieldrin associated with the 1974 EPA ban were extracted to determine if any trends are appearing and how long residues may continue to appear in humans.

Evidence indicates that a dynamic equilibrium exists in the distribution of dieldrin among various tissues in humans, including blood, fat, brain, and liver (*23*). This functional relationship among tissues indicates that the concentration of dieldrin in one tissue is a function of the total amount of dieldrin in the body. Consequently, at least in the case of chronic exposures, the concentration of dieldrin in any tissue may be used as an index of the total body burden of dieldrin. Since dieldrin tends to concentrate in lipids, residue levels in human adipose tissue are frequently used to represent relative total body concentrations.

One of the easiest ways to obtain human fat is to extract milk from lactating women, since human milk contains 3–3½ percent fat. In 1975, EPA obtained 1,436 human milk samples for its *National Study to Determine Levels of Chlorinated Hydrocarbon Insecticides in Human Milk, 1975–76* (*44*). The study was carefully designed to include inhabitants of both rural and urban areas, as well as members of different socioeconomic groups, in order to provide a profile of dieldrin distribution in the United States. Residue levels were calculated on a fat-adjusted basis.

Dieldrin levels were ubiquitous in the human milk samples; 96 percent had residues greater than 1 ppb. The mean value of dieldrin in human milk was 0.164 ppm. These findings correspond closely to the results from the *1974 Survey of Pesticide Residues and Their Metabolites in Humans* (*26*), which showed dieldrin residues averaging 0.150 ppm in human adipose tissue. Thus it appears that fat-adjusted milk residue levels do represent a legitimate measure of total body burden. Dieldrin residues reflect exposure to both aldrin and dieldrin, because aldrin is quickly converted to dieldrin in the human body. Statistics on domestic sales of aldrin and dieldrin between 1960 and 1976 appear in Table 1. Dieldrin residues in adipose tissue from several locales between 1963 and 1977 are listed in Table 2. Studies of aldrin/dieldrin dietary intake between 1964 and 1975 are summarized in Table 3. Figure 1 charts data from all three tables.

Total aldrin and dieldrin sales increased constantly until 1966, then decreased until the EPA ban in 1974. The 1975 dietary intake was about 50 percent of that in 1966, the peak year (Table 3). Yet residues in human tissue have remained astonishingly constant. Thirteen studies from the United States, Canada, England, and Holland show dieldrin levels between 0.150 ppm and 0.220 ppm between 1963 and 1976 (*6, 26, 39, 48*).

Aldrin residues in food increased dramatically in 1975, after decreasing steadily since 1964. This increase may have been a result of the EPA ban itself. Since dieldrin is no longer manufactured in the United States, aldrin is important for registered use as a subsoil termiticide. An increase of aldrin residues in food would be expected to cause a corresponding increase of dieldrin residues in human tissues. However, no significant increase is apparent.

The exact cause of the unchanging concentrations is unknown. In view of the wide fluctuations in sales and dietary intake, simple persistence and biomagnification seem insufficient explanations. Hunter, et al. (*22*), after many years of research, concluded, that "the rates of increase of body burdens progressively decline in an asymptotic manner, the eventual body burdens being a characteristic of a person and his particular

TABLE 2. *Dieldrin residues in human adipose tissue, 1963–76*

Year	No. of Persons Sampled	% Positive	Mean, ppm	Location	Literature Cited
1963			0.15 ± 0.02	USA	(*6*)
1964			0.16	Canada	(*39*)
1966	47		0.21	England	(*39*)
1965–67	146		0.22	USA	(*39*)
1968	11	100	0.17	Holland	(*39*)
1970	200	100	0.20	USA	(*48*)
1970	1,412	97	0.18	USA	(*26*)
1971	1,615	99	0.22	USA	(*26*)
1972	1,913	98	0.18	USA	(*26*)
1973	1,094	99	0.18	USA	(*26*)
1974	898	99	0.15	USA	(*26*)
1975	779	96	0.17	USA	(*26*)
1976	682	94	0.15	USA	(*26*)

TABLE 3. *Adult dietary intake of aldrin and dieldrin in the United States, 1964–75*[1]

Year	% Positive Food Samples		µg/kg/day			µg/day			Literature Cited
	Aldrin	Dieldrin	Aldrin	Dieldrin	Total	Aldrin	Dieldrin	Total	
1964–65	5.6	18.5	0.01	0.08	0.09	1.0	5.0	6.0	(*11*)
1965–66	3.7	21.3	0.04	0.09	0.13	2.0	7.0	9.0	(*11*)
1966–67	3.3	15.3	0.01	0.05	0.06	1.0	4.0	5.0	(*11*)
1967–68	3.9	15.6	0.01	0.05	0.06	T	4.0	4.0+	(*9, 10*)
1968–69	1.4	25.3	0.0001	0.07	0.0701	T	5.0	5.0+	(*8*)
1969–70	0.8	31.3	0.0006	0.07	0.0706	T	5.0	5.0+	(*8*)
1970–71	—[2]	27.8	—[2]	0.0474	—[2]	—[2]	3.3	—[2]	(*33*)
1971–72	—[2]	23.6	—[2]	0.022	—[2]	—[2]	1.6	—[2]	(*32*)
1972–73	—[2]	27.5	—[2]	0.0470	—[2]	—[2]	3.2	—[2]	(*24*)
1973	0.3	29.7	0.0001	0.0403	0.0404	0.0057	2.8	2.8057	(*17*)
1974	0.3	25.8	0.0001	0.0441	0.0442	0.0064	3.1	3.1064	(*17*)
1975	0.4	23.3	0.0022	0.0387	0.0409	0.1500	2.6	2.7500	(*17*)

[1] All data on total food consumption for 15- to 20-year-old males.
[2] Data not available.

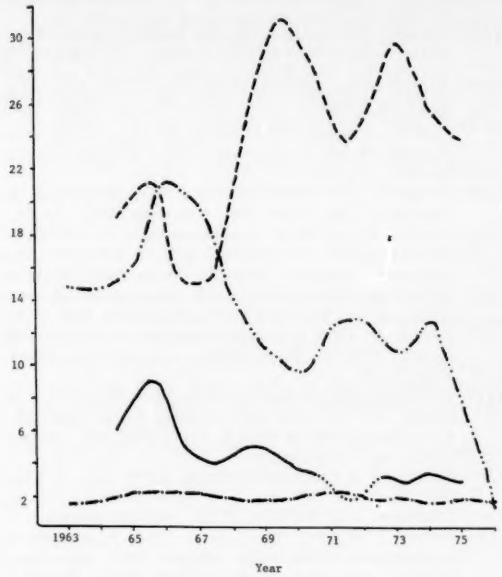

FIGURE 1. *Total domestic sales, percent positive food samples, total United States dietary intake, and human adipose tissue residues of aldrin and dieldrin, 1963–76*

Key: — — Food samples positive for dieldrin (%); — .. — Domestic sales of aldrin and dieldrin (million pounds); —U.S. dietary intake of aldrin and dieldrin (µg/day); . . . . . U.S. dietary intake of dieldrin only (µg/day); . — . — Dieldrin residues in international human adipose tissue (ppm × 10)

daily intake." Keane and Zavon (*25*), studying dogs, also found evidence supporting an "equilibrium hypothesis."

It appears that the concentration of dieldrin in the human body reaches a constant level at which the amount ingested and absorbed equals the amount metabolized and excreted. On the average, the maximum residue level maintained by the body seems to be in the range of 0.150 ppm to 0.220 ppm.

The metabolic process that allows the body to excrete

TABLE 4. *Number and proportion of nursing mothers with fat-adjusted levels of dieldrin greater than 100 ppb by the number of children previously breastfed*[1]

Number of Children Previously Breastfed	Total Number of Mothers	Number	Proportion
0	664	291	0.438
1	463	166	0.359
2	194	63	0.325
3	71	18	0.254
4	24	5	0.208
5 or more	20	5	0.250
TOTAL	1,436–N[2]	548–T[3]	0.382–p

[1] Reference *44*.
[2] N = Total number of mothers involved in the study.
[3] T = Total number of mothers with more than 100 ppb of the chemical in their milk.
[4] $p = T/N$ = proportion of mothers with level of the chemical higher than 100 ppb.

dieldrin when tissue concentrations reach a given level remains unclear. It does seem, however, that the total body burden of dieldrin in some way depends on the degree of obesity of the individual (*25*). A similar homeostatic mechanism has been described for DDT (*35*).

Of course, there may be any number of unidentified factors affecting the storage levels of dieldrin. Several implications have been made, but no definitive conclusions have been drawn. Some evidence suggests that rats fed diets containing DDT store less dieldrin than do control animals (*42*). Similar effects, if they occur in humans, could be counteracting normal decreases in dieldrin levels as environmental exposure to DDT declines in response to the 1972 EPA ban. Hundreds of other chemicals and drugs presently in use could also be affecting residue levels. A highly significant association ($P < 0.001$) between women with low levels of dieldrin in their milk and women who have nursed several children indicates that lactation is one form of organochlorine excretion (Table 4).

It is also possible that the contribution of respiratory intake to total body burden has been underestimated. Average respiratory intake of an adult during the workday is 1.75 cu.m/hr (*12*). Over a 24-hr period,

inhalation could add 1 μg to the daily intake if ambient air levels reached 20 ng/cu.m. Such levels have been recorded in agricultural areas. The possibility that current sampling techniques do not detect some portion of the pesticides in air must also be considered.

A thorough national drinking water survey has not yet been undertaken, so the contribution to dietary intake from water may be underestimated. The exposure of fetuses and very young children to dieldrin could create a whole generation of individuals with high residue levels, despite decreasing exposure during their lifetimes. Finally, one must consider the existence of pesticide reservoirs not yet identified by existing monitoring programs. Routes of transportation, storage, and degradation not yet quantified may exist.

All indications point toward the continuing presence of dieldrin residues in human milk and adipose tissue. Americans are still exposed to dieldrin constantly, through persisting contamination of soil, air, water, and, therefore, food. Evidence suggests that an equilibrium residue concentration is being maintained by most individuals at an average level between 0.150 ppm and 0.220 ppm. The maximum exposure level at which tissue levels will decrease below the equilibrium level is unknown. Further investigation is clearly required in this area as well as into potential influences on residue concentrations. Until the storage and homeostatic processes are fully understood, predictions regarding trends in human tissue residue levels will remain mere speculation.

Acknowledgment

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WATER

Pesticides in Ground Water Beneath Irrigated Farmland in Nebraska, August 1978 [1]

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ABSTRACT

During the 1978 irrigation season, 14 ground water samples were collected in the Central Platte region of Nebraska, an area known to have high nitrate-nitrogen (NO_3–N) levels, and analyzed for the presence of 13 pesticide residues. Atrazine levels ranged from 0.06 μg/liter to 3.12 μg/liter and were correlated to NO_3–N concentrations with a coefficient of r = +0.55. Nitrate-nitrogen concentrations were measured as indicators of deep percolation from irrigated lands and ranged from 17.1 mg/liter to 34.3 mg/liter. Alachlor levels ranged from <0.01 μg/liter to 0.71 μg/liter. The amounts of 2,4-D were indeterminate because of experimental problems. Levels of the herbicides silvex and EPTC were below the limits of detectability. Levels of the organochlorine insecticides endrin, γ-BHC (lindane), dieldrin, DDT and its primary metabolite DDE, heptachlor and its primary derivative heptachlor epoxide, and methoxychlor were all below the detectable limits of 0.005–0.010 μg/liter.

Introduction

Monitoring of pesticides used currently in Nebraska has indicated that trace amounts of atrazine are infiltrating into the shallow ground water beneath well drained irrigated soils in Merrick County (*16*) and in the Platte River bottomland of Hall and Buffalo counties (*10*). In isolated cases, alachlor has also been found in ground water from the Platte River bottomland (*10*). In an agricultural state like Nebraska, where almost the entire population depends on subsurface sources for drinking water, knowledge of possible ground water contamination caused by vertical movements of pesticides through the unsaturated layer is imperative. Thus, the purpose of this study was to measure the levels of selected pesticides in ground water overlain by cropped and irrigated medium-textured silt loam soils having a moderately thick unsaturated layer. Most of the pesticides were selected because of current or past usage, but others were chosen because they have been mentioned in the *Federal Register* (*5*, *6*) in discussions on primary national drinking water regulations.

Ninety-four percent of the irrigated corn acreage in central Nebraska is treated with herbicides. Atrazine is the most frequently used herbicide in the study area, and alachlor is the second most widely used, according to Johnson and Byers (*9*). Alachlor is generally applied at pre-emergent rates of about 0.7 kg/ha as an alachlor/atrazine mixture.

2,4–D is used on less than 10 percent of the irrigated corn acreage and is applied after crop maturity at rates of about 0.5 kg/ha. Water solubility of over 900 μg/liter for both the acid and amine forms of 2,4–D and relatively low sorption characteristics suggest that some infiltration through well drained soils could occur.

EPTC and simazine are used for weed control only where shattercane is a problem. Usually this occurs on less than 3 percent of the corn acreage within the study area.

Silvex is not known to have been used within the study area but was measured because its use may be regulated in the future (*6*).

The γ-isomer of hexachlorocyclohexane (lindane) was one of the earliest used soil insecticides in the study area and was replaced by aldrin and heptachlor. Both

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were used extensively for corn rootworm control until 1962 when emergence of resistant strains reduced their effectiveness. In the soil, aldrin and heptachlor are converted rapidly to dieldrin and heptachlor epoxide, respectively (*13*). The occurrence of dieldrin in ground water beneath Iowa cropland (*14*) and the occurrence of other insecticides in ground water beneath South Carolina cropland (*1, 15*) also prompted the measurement of these insecticides as probable indicators of past contamination.

The foliage insecticide DDT was applied extensively in the study area prior to 1962. Richard et al. (*14*) reported trace amounts of DDE in Iowa well water, and Achari et al. (*1*) and Sandhu et al. (*15*) reported that DDT and its metabolites were the most common pesticide residues in South Carolina ground water.

Methoxychlor and endrin were used either very sparingly or not at all within the study area but they were also mentioned in the *Federal Register* (*5, 6*).

Methods and Materials

SAMPLING

Water samples were collected in an area where NO_3–N concentrations in ground water exceeded 10.0 mg/liter (*7*). The well locations shown in Figure 1 are in an area where a low terrace borders the northern bottomland of the Platter River in central Nebraska. The terrace appears to be flat but does have a very gentle slope of 3.2 m/km, which is also the gradient of the east-northeast-flowing Platte River. The wells that were sampled are predominantly in irrigated areas of silt loam soils where corn is grown. Soils range from 1 m to 2 m thick and are underlain by an unsaturated layer composed of alluvial sand and gravel. Depth to ground water ranges from about 6 m on the southern edge of the terrace to slightly more than 10 m (*4*). Well depths are 20–23 m, which is near the boundary between the upper Pleistocene sand and gravel and the blue clayey silt. None of the wells penetrated into the Ogallala Formation, which lies beneath the clayey silt.

On August 1, 1978, water samples for NO_3–N and pesticide analyses were collected from the 14 irrigation well sites shown in Figure 1. If the well was not already being pumped at the time of sampling, about 20,000 liters of water were exhausted before the sample was collected.

ANALYSES

For the determination of NO_3–N, samples were collected in acid-cleaned, polyethylene bottles and were chilled until they were returned to the laboratory for filtration through 0.45-μm filters; filtered samples were refrigerated at 4°C until the time of analysis. Concentrations were determined by the automated cadmium reduction method of the American Public Health Association (*2*). The waters for pesticide analyses were not filtered. All pesticides were extracted from 4-liter samples, using XAD–2 resin, by the procedure of Junk et al. (*11*). Samples to be analyzed for the phenoxy herbicides were acidified in the field to a pH of 2.0 prior to extraction. Pesticide residues were analyzed by electron-capture gas chromatography and different polarity columns with periodic confirmation by GC/MS.

Results and Discussion

Detectable concentrations of atrazine occurred in all ground water samples, as shown in Table 1. Concentrations ranged from 0.06 μg/liter to 3.12 μg/liter. The significant correlation of +0.05 between atrazine and NO_3–N suggests that where the levels of NO_3–N are higher in the infiltrating water there are correspondingly higher amounts of atrazine. In an earlier study of another Central Platte area having very shallow depth

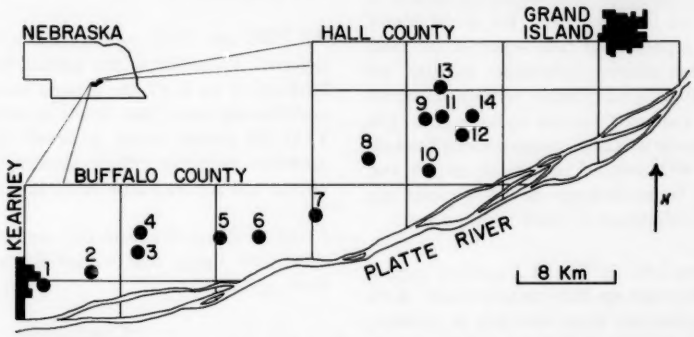

FIGURE 1. *Locations of the irrigation wells, sampled during August 1978 for pesticide analyses, in the Central Platte region, Nebraska, where the NO_3–N levels are >10 mg/liter and the sampled wells are assumed to be representative of over 1000 wells located within the study area.*

TABLE 1. *Pesticide and NO_3–N levels in ground water collected from irrigation wells in Central Nebraska, August 1978*[1]

Well No.	Legal Location[2]	Soil Texture[3]	Unsaturated Thickness, m	NO_3–N, mg/liter	Atrazine µg/liter	Alachlor µg/liter
1	8N–15W–5BB	si l	8.0	23.5	1.23	<0.010
2	9N–15W–35BC	si l	8.3	32.9	3.12	0.071
3	9N–14W–29BA	si l	9.3	34.0	0.82	<0.010
4	9N–14W–17CC	si cl l	8.3	34.4	1.41	0.018
5	9N–13W–19BB	si cl l	6.0	19.8	0.58	<0.010
6	9N–13W–21AB	l	6.0	10.9	0.16	<0.010
7	9N–12W–7CC	l	5.6	17.3	0.06	<0.010
8	10N–12W–27AC	si l	10.0	17.0	0.84	<0.010
9	10N–11W–8CC	si l	9.7	19.0	0.19	<0.010
10	10N–11W–32BB	si cl l	6.7	15.0	0.47	<0.010
11	10N–11W–9CB	si l	9.7	21.9	0.38	<0.010
12	10N–11W–15CD	si l	8.3	31.3	0.21	<0.010
13	11N–11W–33CC	si cl l	10.6	20.5	0.06	<0.010
14	10N–11W–11CD	si l	8.6	17.1	1.00	<0.010

[1]None of the other 11 pesticides searched for in these well waters were found above the detection limits in µg/liter of: 0.001 for lindane; 0.002 for heptachlor and heptachlor epoxide; 0.005 for endrin and dieldrin; 0.005–0.010 for DDE and methoxychlor; and 0.010 for DDT, EPTC, 2,4-D, and silvex.
[2]See Figure 1 for approximate geographical locations.
[3]si = silt; cl = clay; l = loam.

(<3 m) to ground water (*16*), the significant correlation between NO_3–N and atrazine appeared to be related to differences in soil texture such that higher concentrations were observed generally for ground water overlain by coarse-textured soils. The present data indicate that substantial amounts of atrazine move also in medium-textured soils and this vertical movement is not halted by unsaturated layers of 5.6–m to 10.6–m thickness.

Atrazine levels in ground water collected during the late summer of an irrigation season have been shown previously to be associated with vertical transport of atrazine applied during the same season (*10*). This being the case, an estimated 1 percent of the applied atrazine migrated vertically through the vadose zone (zone of aeration) to the ground water during the 1978 irrigation season. This estimate is based on an average atrazine concentration of 0.75 µg/liter, an average saturated zone thickness of 10.7–m depth with 20 percent porosity, and an average atrazine application rate of 0.75 kg/ha (*9*). Although this leakage estimate is close to that obtained by Lavy (*12*) for sandy loams in Nebraska, it is higher than that reported by Hall and Hartwig (*8*) for atrazine infiltration through silt loams in Pennsylvania. This greater vertical transport of atrazine in this area may be due to irrigation. The reason for the scatter in atrazine concentrations beneath irrigated farmland with soils of equivalent texture and similar unsaturated layer thickness is not obvious but may well be due to differences in water management.

Arachlor levels above 0.01 µg/liter in 2 samples suggest alachlor transport through medium-textured soils. Both samples contained relatively large amounts of atrazine, indicating that a formulation of the two compounds had been used on the upgradient fields.

The 2,4–D in these irrigation well waters could not be determined accurately due to some chromatography problems. However, all of the present data show 2,4–D levels below the detection limit of 0.05 µg/liter. This low level probably reflects limited usage in the study area and rapid degradation rather than poor transport, since Asmussen et al. (*3*) report 2,4–D to be one of the more mobile herbicides.

Concentrations of the herbicides EPTC and silvex in ground water were less than 0.01 µg/liter and 0.005 µg/liter, respectively. No simazine was detected even in waters that contained the highest levels of atrazine. This observation is consistent with the very limited use of these three herbicides within the study area.

Levels of γ-BHC, dieldrin, heptachlor, and heptachlor epoxide were below the limits of detection of 0.001, 0.005, 0.002, and 0.002 µg/liter, respectively. These insecticides have not contaminated the ground water, or have been sorbed efficiently to the aquifer sediments, or have degraded over the past 15 years since their use was discontinued.

All DDT and DDE concentrations were less than 0.01 µg/liter. These levels are similar to those reported by Richard et al. (*14*) for ground water in Iowa but are significantly lower than levels reported by Sandhu et al. (*15*) for ground water in South Carolina. The higher amounts probably reflect greater usage of DDT on cotton and soybean crop lands in that area.

Concentrations of endrin and methoxychlor were below detectable limits that varied from 0.005 µg/liter to 0.01 µg/liter.

Conclusions

Concentrations of pesticide residues in the ground water surveyed in the present study are well below the limits given in the interim primary drinking water

standards. The pesticide contamination of the ground water in the heavily irrigated study area in Nebraska is less than that reported for surface waters from New Orleans and Iowa (*14*) and for ground water from South Carolina (*15*).

Measurable amounts of atrazine in all samples, and alachlor amounts greater than 0.010 μg/liter in two samples, indicate that there is some vertical movement of the herbicides used extensively in the study area. The wide range in atrazine concentrations in water under predominantly silt loam soils suggests vertical transport associated with possible differences in water management. Whether this pesticide contamination of the ground water could be reduced or even eliminated with improved management is debatable.

Although the low levels of atrazine in the ground water are not toxic to humans, Wolfe et al. (*17*) report that the presence of atrazine and high amounts of NO_3–N in the ground water may evoke a further complication. They report formation of *N*-nitrosoatrazine in nitrogen-fertilized soils. Thus monitoring of nitrosamines and their probable precursors in the ground water of the Central Platte may be warranted.

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APPENDIX

Chemical Names of Compounds Discussed in This Issue

ALACHLOR	2-Chloro-2',6'-diethyl-*N*-(methoxymethyl)-acetanilide
ALDRIN	Hexachlorohexahydro-*endo,exo*-dimethanonaphthalene 95% and related compounds 5%
AROCLOR 1254	PCB, approximately 54% chlorine
AROCLOR 1260	PCB, approximately 60% chlorine
ATRAZINE	2-Chloro-4-(ethylamino)-6-(isopropylamino)-*s*-triazine
BHC (Benzene Hexachloride)	1,2,3,4,5,6-Hexachlorocyclohexane (mixture of isomers)
CHLORDANE	Technical: 60% octachloro-4,7-methanotetrahydroindane and 40% related compounds
2,4-D	2,4-Dichlorophenoxyacetic acid
DDE	Dichlorodiphenyldichloroethylene (degradation product of DDT)
DDT	Dichloro diphenyl trichloroethane. Principal isomer present (*p,p'*-DDT; not less than 70%): 1,1,1-trichloro-2,2-bis(*p*-chlorophenyl)ethane
DIELDRIN	Hexachloroepoxyoctahydro-*endo,exo*-dimethanonaphthalene 85% and related compounds 15%
ENDRIN	Hexachloroepoxyoctahydro-*endo,endo*-dimethanonaphthalene
EPTC	*S*-Ethyl dipropylthiocarbamate
HCB	Hexachlorobenzene
HEPTACHLOR	Heptachlorotetrahydro-4,7-methanoindene
HEPTACHLOR EPOXIDE	1,4,5,6,7,8,8-Heptachloro-2,3-*epoxy*-3a,4,7,7a-tetrahydro-4,7-methanoindan
LINDANE	*Gamma* isomer of benzene hexachloride (BHC)
METHOXYCHLOR	2,2-Bis(*p*-methoxyphenyl)-1,1,1-trichloroethane 88% and related compounds 12%
METHYL PARATHION	*O,O*-Dimethyl *O*-*p*-nitrophenyl phosphorothioate
MIREX	Dodecachlorooctahydro-1,3,4-metheno-1*H*-cyclobuta[cd]pentalene
NONACHLOR	1,2,3,4,5,6,7,8,8-Nonachlor-3a,4,7,7a-tetrahydro-4,7-methanoindan
OXYCHLORDANE	1-*exo*-2-*endo*-4,5,6,7,8,8a-Octachloro-2,3-*exo*-*epoxy*-2,3,3a,4,7,7a-hexahydro-4,7-methanoindene
PCBs (Polychlorinated Biphenyls)	Mixtures of chlorinated biphenyl compounds having various percentages of chlorine
SILVEX	2-(2,4,5-Trichlorophenoxy)propionic acid
SIMAZINE	2-Chloro-4,6-bis(ethylamino)-*s*-triazine
TDE	Dichloro diphenyl dichloroethane (1,1-dichloro-2,2-bis(*p*-chlorophenyl)ethane, principal component)
TOXAPHENE	Technical chlorinated camphene (67–69% chlorine)

The *Pesticides Monitoring Journal* welcomes from all sources qualified data and interpretative information on pesticide monitoring. The publication is distributed principally to scientists, technicians, and administrators associated with pesticide monitoring, research, and other programs concerned with pesticides in the environment. Other subscribers work in agriculture, chemical manufacturing, food processing, medicine, public health, and conservation.

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